DISEASES *of the* CHEST

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Bacteriologic Examination of Tissues Surgically Removed as an Aid in the Diagnosis of Diseases of the Chest*

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With the marked progress that has been made in recent years in the treatment of certain types of pulmonary disease by means of chemical and antibiotic agents and with the advances that have been made in the successful eradication of others by means of thoracic surgery, it becomes increasingly important to determine as far as possible the underlying etiology of a pulmonary lesion so that the most satisfactory method of treatment can be employed.

The importance of establishing an early and positive diagnosis in all cases of pulmonary disease is especially manifest when it is realized that carcinoma of the lung and tuberculosis have the ability to mimic practically any type of pulmonary disease. Prolonged periods of observation might often enable one to arrive at an eventual determination of the character of the lesion, but in dealing with the possibility of carcinoma and tuberculosis, time becomes a matter of greatest essence.

With the improvement that has taken place in recent years in the field of thoracic surgery, it is often advisable and wise to carry out prompt exploratory thoracotomy in cases of indeterminate pulmonary disease. Such a procedure will frequently reveal unexpected and interesting findings. It is important in cases of this type, if the full value of the operation is to be attained, that

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the tissue or secretion which is removed be examined by a competent pathologist.

Since time immemorial, pathologic processes have been shrouded with varying degrees of mysticism. With the development of the concept of cellular pathology and with an appreciation of physiologic mechanisms a more rational approach has been made to the understanding of disease. However, cellular pathology rests exclusively on a morphologic basis, and interpretations and opinions concerning such pathologic processes as may be observed grossly and microscopically are limited to one's ability to appreciate and recognize varying degrees of morphologic change. In the field of oncology various procedures have been developed by which one may not only recognize the nature of the tumor but may also express an opinion as to the rapidity of growth with a view to prognosticating the outcome of the disease.

With the growth of the cellular theory of pathology, technics have been developed by which the tissues to be examined are either killed by some suitable fixing agent, such as formalin or mercuric chloride, or are frozen in the fresh state so that an immediate histologic preparation may be made. The former method kills not only all the tissue cells but also the microbic agents which may be the cause of the lesion. In the second method, the handling of tissues preparatory to making the frozen section results in contamination so that a suitable bacteriologic examination may be impossible should the histologic study reveal the desirability of such examination.

Because many different infectious diseases produce histologic patterns which grossly simulate one another, it becomes the duty and the responsibility of the pathologist to be prepared to establish the nature of the etiologic agent inducing any inflammatory processes which he considers unlikely to be a part of, or related to neoplastic changes. While modern pathologists recognize the importance of consulting with bacteriologists or of making their own bacteriologic examinations, concerning certain inflammatory processes it should be emphasized that a bacteriologic examination should supplement, not replace, the histologic study, since malignant lesions may be secondarily infected and may simulate an infectious granuloma. We have found it of value to handle all thoracic surgical specimens and material for biopsy in such a manner that satisfactory bacteriologic examinations may be made in the event an inflammatory process is encountered; thus we avoid subjecting the patient to the expense and inconvenience of a repeated biopsy. The importance of such supplementary aid to the clinician and pathologist is exemplified in the following cases.

REPORT OF CASES

Case 1: A woman forty-two years of age who resided in Oklahoma, presented herself at the Mayo Clinic for examination on August 19, 1946. She gave a history of rheumatic pains which had been present for four years. Her primary reason for seeking medical attention at that time was to learn the nature of a questionable lesion in the pelvis.

At the time of examination the patient was found to have a small uterine fibroid. The fibroid was of such size and character that surgical intervention did not seem indicated. During the course of a general examination a roentgenologic examination of the chest revealed a discoid or globular mass lying just subcostal to the posterior portion of the tenth rib on the left side (Fig. 1). This was suggestive, to the roentgenologist, of either a benign tumor or an intercostal neurofibroma. Results of examination of the sputum for acid-fast organisms were reported as negative. The urinalysis, hemoglobin determination, erythrocyte and leukocyte counts, and determination of the sedimentation rate gave values which were within normal limits; the reaction to flocculation tests for syphilis was negative.

From the history and the information at hand, it was felt that the condition might be a hamartoma although the possibility of malignancy could not be excluded. Exploratory thoracotomy seemed advisable in this case and the patient was so advised. At operation on October 29, 1946, a firm, rounded tumor mass was found situated near the periphery of the lower lobe of the left lung. This mass was excised. The specimen removed was reported by the pathologist as having the gross and microscopic appearance suggestive of healed primary tuberculosis (Fig. 2). Material from the tumor mass was injected into guinea pigs in order to determine definitely the underlying etiologic agent. Much to our surprise, when the animals were examined at necropsy eight weeks later they were found to be infected with Coccidioides immitis and there was no evidence of tuberculosis. Because of this report, the original tissue was re-examined and additional sections were taken from the tumor mass; at this time numerous small spherical bodies were recognized. Some of these bodies appeared to be in the budding form, but none could

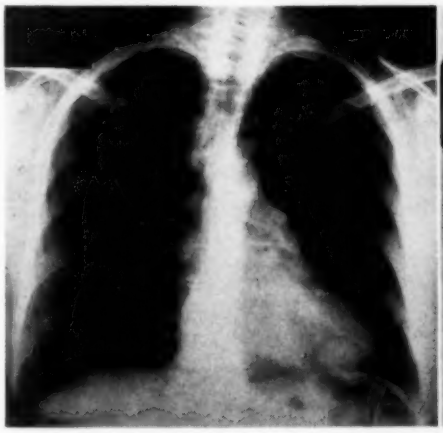

FIGURE 1

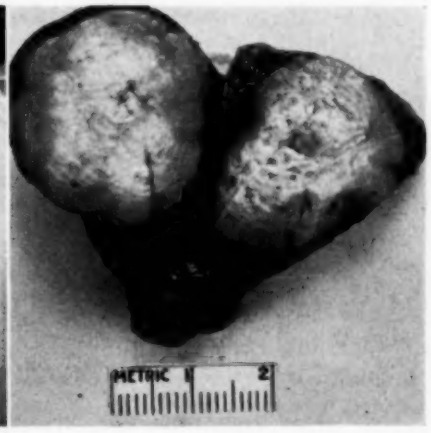

FIGURE 2

Fig. 1 (case 1): Circumscribed tumor mass in left base.
Fig. 2 (case 1): Gross appearance of tumor removed.

be found which had the endosporulating spherules characteristic of coccidioidomycosis.

In retrospect, the diagnosis of this lesion might have been made if a skin test for coccidioidomycosis had been carried out. However, the clinical history and the fact that the patient came from a part of the country where coccidioidomycosis is not endemic did not arouse our suspicion of a coccidioidomycotic infection. The case does emphasize the importance of keeping the possibility of coccidioidomycosis in mind in all cases of pulmonary infiltration. Although surgical intervention is not recommended as a routine procedure in coccidioidomycosis, it is of interest that this patient made very satisfactory progress after the removal of the coccidioidal nodule from her lung. It is, of course, problematic whether the improvement in her general rheumatic pains can be justifiably ascribed to the removal of this nodule.

Case 2: A woman forty-six years of age who came from Minnesota was admitted to the clinic September 18, 1946. Her chief complaint was that of cough and expectoration. She stated that her difficulty began after an attack of pneumonia in 1919. Since that time she had suffered from a chronic cough and had been subject to frequent pulmonary infections. For the past five years she had coughed up a great deal of purulent secretion; the quantity varied from a half teaspoonful to a half cupful in twenty-four hours. After overexertion the sputum had at times been blood streaked. During the past year the patient had noted an increasing degree of dyspnea associated with exertion.

On physical examination the only essential findings were a slight

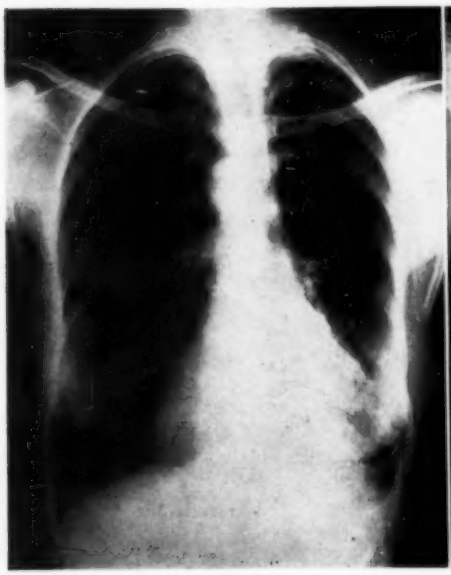

FIGURE 3

FIGURE 4

Fig. 3 (case 2): Contraction of right lower lobe.
Fig. 4 (case 2): Bronchiectasis of right lower and middle lobes.

limitation in movement of the right side of the chest on respiration and a few coarse rales heard over the right lower and middle lobes. When the patient was tipped over she expectorated a characteristic bronchiectatic type of sputum. Roentgenologic examination of the chest was reported as showing probable contraction of the right lower lobe (Fig. 3). Sputum examination did not reveal acid-fast organisms. Bronchoscopic examination was performed and a large amount of purulent secretion was found coming from the bronchi of the right lower and middle lobes. The mucous membrane of the bronchus of the right lower lobe was markedly inflamed. No evidence of a tumor or stenosis of the bronchus was seen. Bronchograms were made the following day; these showed evidence of bronchiectasis involving the right lower and middle lobes (Fig.4).

A diagnosis of bronchiectasis involving the right lower and middle lobes was made and operation was advised. After preoperative nebulization with penicillin the patient was operated on on October 26, 1946. The middle lobe was found to be completely atelectatic. A right middle and lower lobe lobectomy was performed. On gross and microscopic examination the pulmonary tissue removed showed the characteristic appearance of bronchiectasis with associated pneumonitis (Fig. 5a). Since pneumonitis is a common finding in bronchiectasis, in this case it was not thought necessary to carry out further pathologic or bacteriologic studies to determine the etiology of this lesion.

After the operation collapse of the right upper lobe developed. In spite of repeated bronchoscopic aspirations the right upper lobe failed to reexpand. It became necessary to perform a right upper lobe lobectomy in December 1946. The gross and microscopic appearance of the right upper lobe was the same as that found in the right lower and middle lobes at the time of their removal, except that the degree of bronchiectasis was less marked (Fig. 5b). The material removed at this time was subjected to bacteriologic examination and was found to contain Pasteurella of the animal variety. Empyema of the right side of the chest developed after the right upper lobe lobectomy; drainage was obtained by open operation. Although the Pasteurella in this case was found to be resistant to streptomycin in vitro, the antibiotic agent was instilled into the empyema pocket with apparent benefit.

There might be some question as to whether the Pasteurella infection in this case was primary or secondary in nature. In any case, it is unusual that the involvement of the upper lobe should have occurred after the lower and middle lobe lobectomy and that the pathologic process found in all three lobes was identical in nature. It would seem that the Pasteurella was the underlying etiologic factor in this case.

Case 3: A man thirty-two years of age whose home was in Mexico came under our care on September 26, 1946, stating he had cancer of the lung. He said that he had enjoyed good health until May 1946, when he first noticed a sharp pain in the left side of the chest. The pain was not related to respiration and lasted but three days. With the onset of the chest pain he noted an elevation of temperature to 40.5° C. Roentgenologic examination of his chest at that time was reported as disclosing evidence of an area of infiltration which involved the left lower lobe

FIGURE 5 (case 2): (a) Section of right lower lobe (x3).
(b) Section of right upper lobe (x3.5).

and which led to a diagnosis of congestion of the left lower lobe. The patient was treated with injections of penicillin without apparent improvement. His temperature remained elevated for twenty-one days. Since the onset of illness the patient had lost weight. He had had a very slight, unproductive cough. Because the roentgenograms of the chest failed to show evidence of any improvement, carcinoma of the lung was suspected and the patient was referred to us for treatment.

The patient was found to be a well-developed, healthy-appearing man with normal blood pressure, pulse and temperature. Physical examination of the chest gave essentially negative results. Urinalysis, hemoglobin determination, erythrocyte and leukocyte counts and differential count gave results which were within normal limits and the reaction to the flocculation test for syphilis was negative. It was impossible to obtain sputum for culture or microscopic examination for tuberculosis or for examination for malignant cells. A roentgenogram of the chest made on September 27, 1946, showed evidence of a lesion in the left lower pulmonary field which was partially obscured by the left side of the heart (Fig. 6). The lesion was considered by the roentgenologist to be probably a hamartoma. Bronchoscopy was done and gave entirely negative results. The history of pulmonary difficulty of four months' duration, with loss of weight and a persistent nonresolving chest lesion as seen on roentgenologic examination, made the diagnosis of carcinoma very probable.

Exploratory thoracotomy was advised. The patient was operated on on October 26, 1946, and a lesion was found situated in the inferior portion of the left upper lobe which appeared to be inflammatory in nature. It was adherent to the pericardium as well as to the chest wall. A left upper lobe lobectomy was performed. The specimen removed was reported by the pathologist as being a granulomatous inflammatory mass, possibly a lesion of coccidioidomycosis (Fig. 7). However, no endosporulating spherules were found although budding forms were present, suggesting the possibility of blastomycosis. Bacteriologic studies of the tissue revealed large numbers of Coccidioides immitis; no blastomycetes were recovered in the culture, although their presence had been sug-

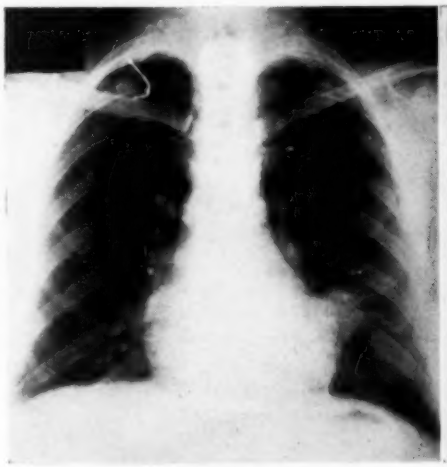

FIGURE 6

FIGURE 7

Fig. 6 (case 3): Lesion in left lower pulmonary field.
Fig. 7 (case 3): Mass removed at operation.

gested by the appearance of the budding forms in the microscopic examination. Coccidioidin skin tests were done after operation, and the results were found to be positive.

In this case, the possibility of coccidioidomycosis should undoubtedly have been suspected before operation; however, this disease was not suspected and it was necessary for the bacteriologist to have special studies carried out in order to permit determination of the exact cause of the patient's difficulty. This patient did very well after operation.

Case 4: A woman forty-nine years of age came to the clinic for examination on March 3, 1947. She had always enjoyed good health until February 19, 1946. At that time she suffered from a sore throat which she stated was not severe and which lasted but a few days. Four days later a red rash developed on the left shin, followed in a few days by similar lesions on both wrists and hands. The patient described these lesions as looking like burns. The onset of the skin lesions was associated with an elevation of temperature to 102° F. The patient was hospitalized in her home community for three weeks, during which time the skin lesions disappeared and the temperature returned to normal. At the end of her period of hospitalization, her abdomen began to enlarge and she suffered from increasing dyspnea on exertion, orthopnea and severe edema of the lower extremities. She was again hospitalized and kept in an oxygen tent for seven weeks. She stated that during this period she had generalized edema with daily fever, periods of delirium and a pleural

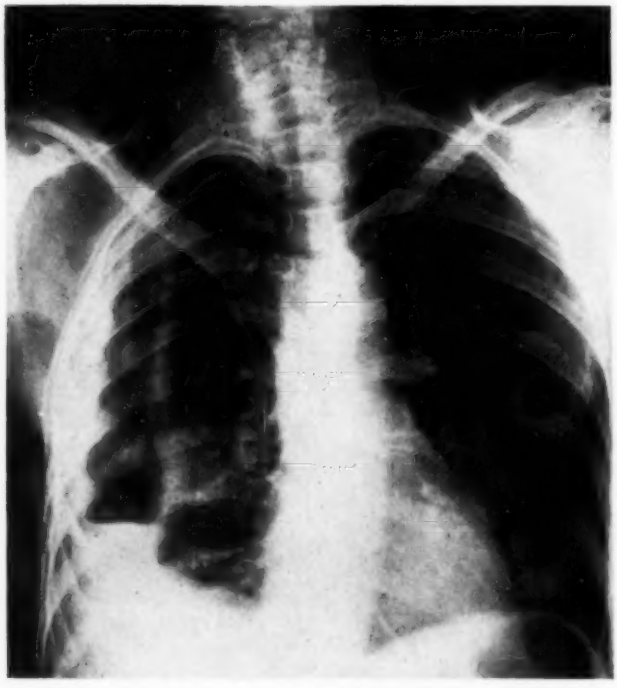

FIGURE 8 (case 4): Encapsulated empyema and thickening of pleura.

effusion on the right side. Aspiration was performed two times and both times the fluid removed was reported as clear. By June 1, 1946 most of the afore-mentioned symptoms had subsided. On December 10, 1946 she again suffered from a sore throat with elevation of temperature to 103° F., a cough and expectoration of small amounts of yellowish sputum; hemoptysis did not occur. The cough persisted for one week and then disappeared. However, a low-grade fever persisted until February 19, 1947. During this entire episode the patient lost forty-five pounds (20.4 kg.).

At the time of our examination on March 3, 1947 the patient was very thin, weighing but a hundred pounds (45.4 kg.); she obviously was ill. On physical examination the right side of the chest was found to be retracted, with lagging of this side on respiration; there was dullness to flatness to percussion over this side with diminution of breath sounds. Roentgenographic examination of the chest was reported as revealing an encapsulated empyema on the right side with rather marked thickening of the pleura; a fluid level was present opposite the seventh interspace posteriorly (Fig. 8). Urinalysis, hemoglobin determination, erythrocyte and leukocyte counts and differential count all gave results which were within normal limits. Blood smears showed no significant changes. The sedimentation rate was 70 mm. in one hour (Westergren). Examination of the sputum for acid-fast organisms gave negative results.

A diagnosis of empyema of the right side of the chest was made and

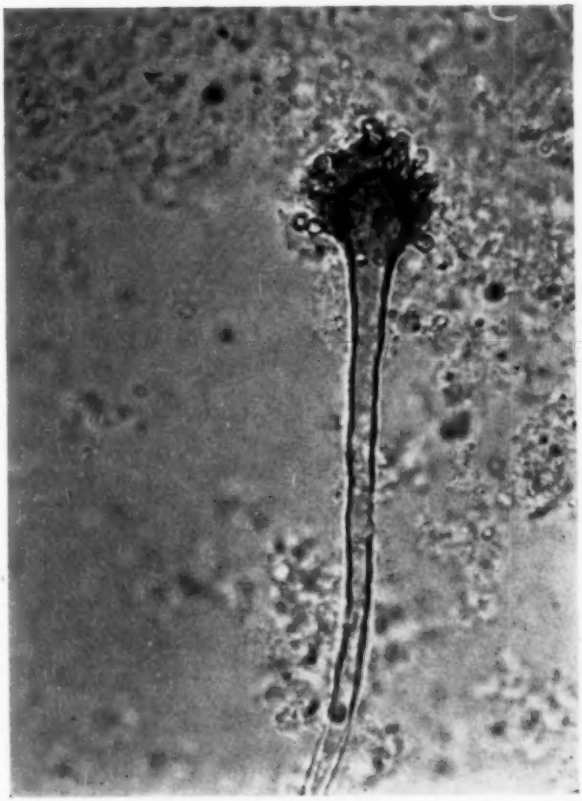

FIGURE 9 (case 4): Direct smear of tissue showing sulfur granules (x720).

a diagnostic aspiration was carried out on March 5, 1947, when 50 cc. of thick greenish gray purulent material was aspirated. This material was examined for acid-fast organisms and for sulfur bodies with negative results. The patient was operated on on March 7, 1947, the surgeon carrying out resection of four ribs with decortication of the right lung for chronic empyema. Multiple bronchial fistulas were noted at the time of operation. A moderate amount of pus was found in the pleural cavity; this material contained multiple small, yellowish sulfur bodies which the surgeon considered to be diagnostic of Actinomyces. Careful bacteriologic studies of the granules, however, showed them to be composed entirely of large, branching filaments which were finally identified as Aspergillus fumigatus (Fig. 9). Results of studies on guinea pigs were negative for tuberculosis. No other microbic agent was ever obtained in cultures taken from the empyema cavity and it was therefore concluded that the pulmonary lesion was that of aspergillosis. The patient was treated with large doses of penicillin after drainage of the empyema cavity; she showed marked improvement in general health.

Case 5: A man thirty-four years of age who resided in southern California, first came under our observation on September 25, 1946. A month before admission he had first suffered from a dull headache associated with soreness in the back of the neck. He also noted some pain in the right upper part of the chest on deep respiration. He became apprehensive that his condition might be due to poliomyelitis, as there were a number of cases of this disease in his neighborhood at the time. A roentgenogram of the chest made by his family physician showed evidence of a lesion in the right lower lobe. He was found to have a temperature of 100° F. He was placed in the hospital and penicillin was started along with sulfadiazine. The sulfadiazine was soon discontinued because of the development of a generalized rash. At the end of a week the patient felt considerably better but the pain in the chest persisted.

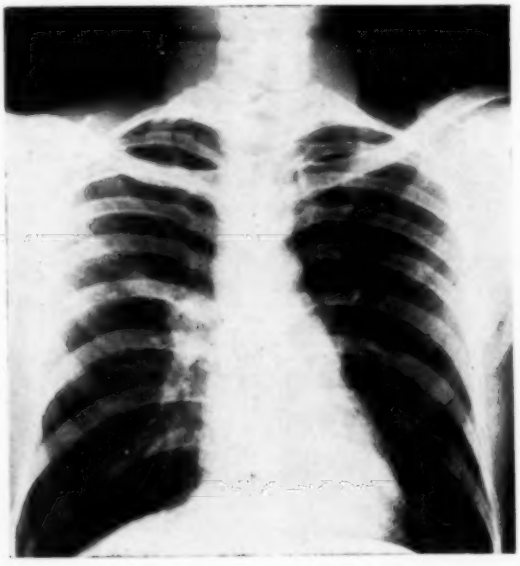

FIGURE 10 (case 5): Lesion in right lung at level of third rib anteriorly. Note cavity.

During the period of hospitalization eight examinations of sputum were carried out and all were reported negative for tubercle bacilli. As the chest lesion seen on roentgenologic examination persisted, a diagnosis of carcinoma was suspected and the patient was referred to the clinic for further study.

At the time of admission to the clinic the patient's only complaint was that of pain in the thoracic part of the spinal column. The patient was a well-appearing, well-developed young man. Physical examination of the chest failed to reveal anything of diagnostic importance. On palpation of the neck a hard gland was found in the right supraclavicular region. The roentgenogram of the chest showed a lesion to be present in the right lung at the level of the third rib anteriorly, with associated fibrosis extending to the second interspace anteriorly (Fig. 10). It was felt that the possibility of tuberculosis must be considered. The sedimentation rate was 53 mm. in one hour (Westergren). Examination of the sputum did not reveal acid-fast organisms. Other laboratory tests were all found to give essentially negative results.

From the history it was believed that the patient's pulmonary lesion was probably on an inflammatory basis and the possibility of a tuberculous infection was strongly entertained. The supraclavicular lymph node was removed for biopsy and on microscopic examination revealed, both in the fresh frozen and the fixed sections, the histologic picture

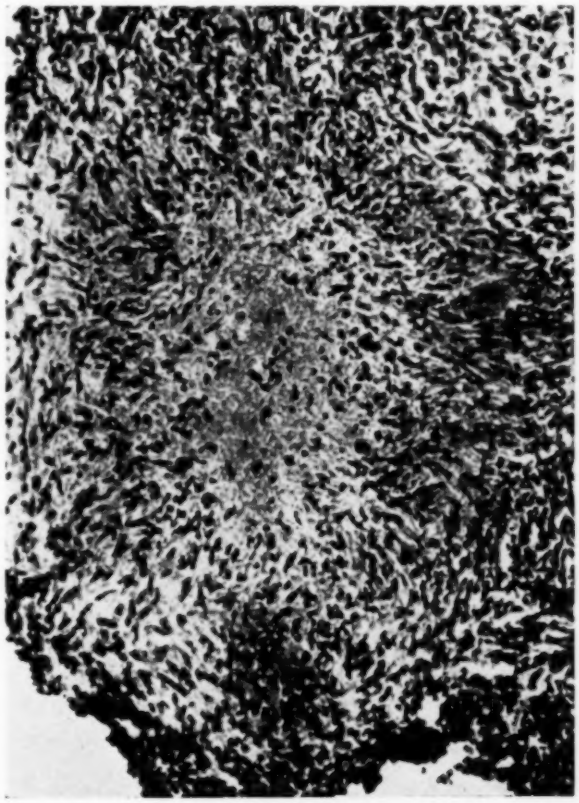

FIGURE 11 (case 5): Section of supraclavicular lymph node. Histologic picture is that of tuberculosis (x160).

of tuberculosis (Fig. 11). Stains of the tissue for acid-fast organisms failed to reveal the presence of any such organisms. In guinea pigs inoculated with material from the lymph node, lesions developed which were suggestive of tuberculosis at the time the lesions were examined at necropsy eight weeks after inoculation. Further studies of the tissue, however, showed no acid-fast organisms to be present, but there were many endosporulating spherules characteristic of Coccidioides immitis (Fig. 12). This was proved to be the etiologic agent by additional cultures and animal studies.

Here again we have striking evidence of the importance and value of bacteriologic study of tissue which, on microscopic examination, is not entirely diagnostic. It emphasizes the importance of close co-operation between the pathologist and the bacteriologist. Perhaps in this case the possibility of coccidioidomycosis should have been suspected as the patient came from California.

Case 6: A young man twenty-three years of age had always enjoyed good health until three years before coming under our observation; at the onset of illness he first had started to lose weight and to fatigue easily. He felt especially exhausted at the end of a hard day's work. During the next four months he lost fifty pounds (22.7 kg.) and it was necessary for him to retire from his work and to remain at rest. Two

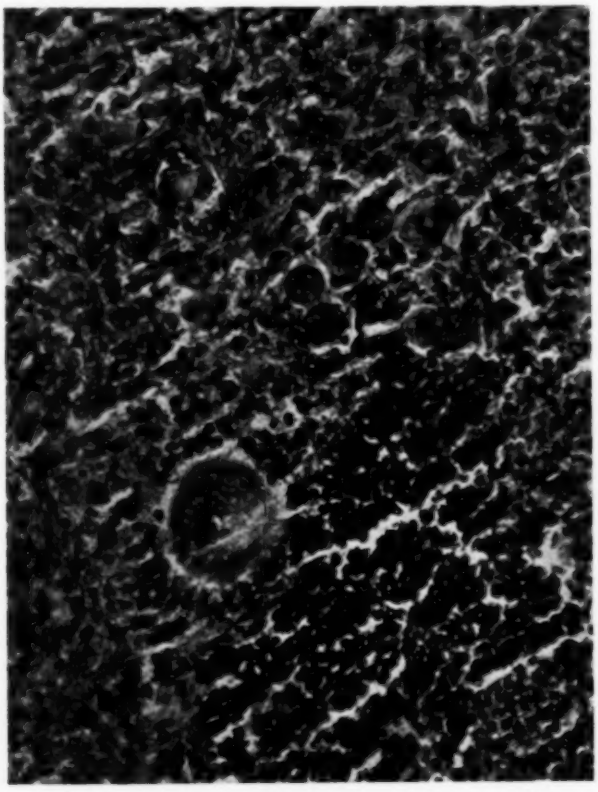

FIGURE 12: Endosporulating spherules characteristic of Coccidioides immitis (x255).

months later he experienced profuse night sweats for several weeks and began to run a low-grade fever. A year after the onset of his difficulty he had seemed to improve and again had returned to work. A month after his return to work a sore spot in the pharynx and swollen, tender, bleeding gums developed. This was diagnosed as trench mouth for which he received treatment without much success. A roentgenogram of the chest was made at the time that the sore mouth developed, and this was reported as showing a clouding of the central portions of both lungs; a diagnosis of tuberculosis was suspected. He was sent to a tuberculosis sanatorium, where he remained for three months. At the end of that time he was discharged because they were unable to confirm the diagnosis of tuberculosis. During the next year the patient remained at rest but still continued to feel fatigued, weak and short of breath on exertion, and had a sore mouth.

At the time of our examination the patient was found to be an undernourished young man who was obviously ill. It was noted that he had several raw areas on the right side of the throat and in the back of the mouth. The gums were swollen and boggy and tended to bleed on manipulation. The remainder of the physical examination revealed nothing of diagnostic importance. Roentgenographic examination of the chest disclosed an extensive pulmonary fibrosis which was much more marked in the middle third of both lungs, with compensatory emphysema in the bases and apices. The roentgenologist thought that the possibility of a fungus infection or sarcoidosis should be excluded (Fig. 13). Examination of the sputum for acid-fast bacilli gave negative results. The sedimentation rate was 65 mm. in one hour (Westergren). The reaction to the tuberculin test was reported as negative. Special laryngologic exam-

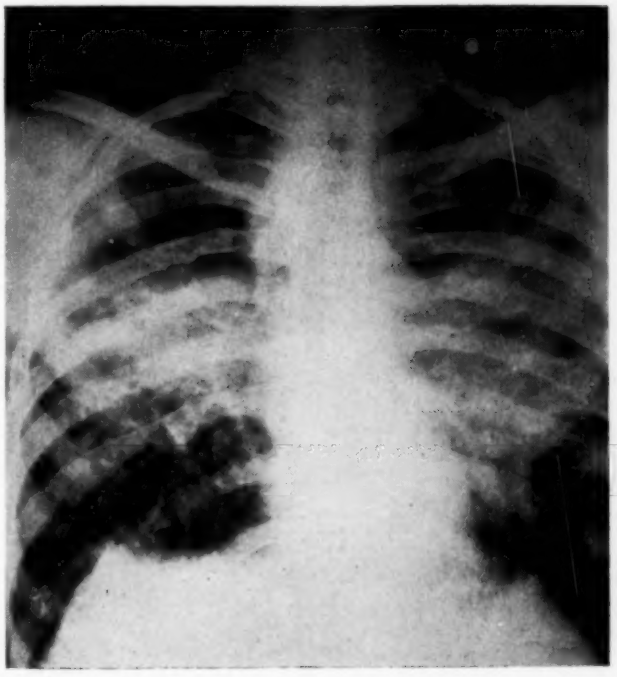

FIGURE 13 (case 6): Marked pulmonary fibrosis and compensatory emphysema.

ination revealed a granulomatous lesion 1.5 cm. in diameter on the right side of the soft palate near its posterior border. There was another lesion of about the same size a little farther forward near the junction of the hard and soft palates on the right side. Another was seen in the left lower alveolobuccal fold opposite the cuspid region, and a fourth involved almost the entire left side of the floor of the mouth. There was also a small lesion involving the right tip of the epiglottis. A specimen was removed from these lesions for biopsy which on microscopic examination was reported as showing evidence of tuberculosis. However, acid-fast organisms could not be demonstrated in the lesions and guinea pigs inoculated with the material failed to show evidence of tuberculosis. Special bacteriologic studies however revealed the presence of Histoplasma capsulatum (Fig. 14).

SUMMARY

Obviously, it should be our cherished goal to try to establish a positive clinical diagnosis in every case of pulmonary disease. Careful and diligent use must be made of the many useful tests and examinations that are available for this purpose. The close co-operation of the roentgenologist, bronchoscopist, bacteriologist and other specialists is highly important. Despite all this, there will remain a goodly number of patients suffering from pulmonary disease in whom a positive diagnosis cannot be arrived at. It is often unwise, in a case of this type, to procrastinate too long with periods of observation or with tests that require many weeks for completion, because of the danger that the lesion may be malignant in nature, thereby requiring prompt treatment if it is

FIGURE 14 (case 6): Histoplasma capsulatum (x950).

to be eradicated successfully. In properly selected cases of this type, exploratory thoracotomy is often indicated; the pathologist can often furnish a prompt explanation of the underlying cause of the pulmonary lesion. In cases in which the cause is uncertain, bacteriologic studies of the tissue should be employed, as often useful information which might otherwise escape detection may be obtained by this method.

RESUMEN

Naturalmente que nuestro propósito debe ser el tratar de establecer un diagnóstico clínico positivo en todo caso de enfermedad pulmonar. Se deben emplear con cuidado y diligencia las muchas pruebas y exámenes valiosos disponibles para este propósito. La íntima cooperación entre el roentgenólogo, broncoscopista, bacteriólogo y otros especialistas es sumamente importante. A pesar de todo esto, quedará un número crecido de pacientes que padecen enfermedades pulmonares en los que no se puede hacer un diagnóstico positivo. En casos de este tipo, a menudo no es prudente dilatar por mucho tiempo con períodos de observación o con pruebas que requieren muchas semanas para completarse, debido al peligro de que la lesión sea de naturaleza maligna y que, por consiguiente, exiga pronto tratamiento para erradicarla con buen éxito. En casos de este tipo, propiamente seleccionados, se indica frecuentemente la toracotomía exploratoria, pues a menudo el patólogo puede suministrar una pronta explicación de la causa de la lesión pulmonar. En casos en los que la causa es incierta deben emplearse estudios bacteriológicos del tejido, pues mediante este método con frecuencia se puede obtener información útil que de otra manera podría pasarse por alto.

Discussion

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We are indebted to Dr. Moersch for bringing us the experience of his group in handling pathologic specimens so that they may also be studied bacteriologically. This presentation is timely for two reasons; first, because coccidioidomycosis is being encountered by some of us who had only read or heard of it before the war. This is due to the fact that many of our soldiers were trained in areas where the disease is endemic. Second, because the work of Drs. Christie and Peterson has added Histoplasmosis to the list of diseases we must differentiate from tuberculosis.

Diseases of the chest are of such complex forms that it is necessary to have the services of many people in order to make a satisfactory diagnosis, and it goes without saying that the various ones concerned must conduct their examinations in such a way as to be of all possible help to each of their colleagues in their part of the examination.

It has been our custom for several years to examine granulomatous and ulcerative tissue obtained by bronchoscopic biopsy both histologically and bacteriologically. Such examinations are not routine but have been of distinct value in several cases.

We also have studied lymph glands obtained by biopsy, both bacteriologically and pathologically, when the differentiation between tuberculosis, Boecks sarcoid, cancer, and Hodgkins disease was to be made. The material so obtained has been sectioned and studied for tubercle bacilli as well as abnormal cells, and at the same time has been inoculated into guinea pigs and cultured.

This method of study resulted in some unexpected diagnoses of Histoplasmosis and coccidioidomycosis and has been helpful in establishing diagnoses of undulant fever.

More recently, in connection with his study of histoplasmosis, Dr. Amos Christie has added an expert mycologist to his staff, and I am privileged to report briefly some of their cases which will be reported in detail by them later.

In one case, a diagnosis of Hodgkins disease had been made by biopsy ten years previously. The patient continued to have the enlarged cervical lymph glands but was little if any worse. Biopsy and histological and bacteriological examination revealed Histoplasma Capsulatum. A second case was that of a young white woman who had an acute infection of her cervical lymph glands which yielded to penicillin but relapsed while under treatment. A biopsy of one of the cervical glands revealed the yeast cells of Histoplasma Capsulatum.

Recently a series of 75 cases of unidentified granulomatous material obtained at autopsy was studied bacteriologically, and four of them yielded definite Histoplasma Capsulatum and four have been tentatively identified as the same organism.

Another case, a child that died of dysentery just before I left Nashville, was autopsied and material obtained from the intestines was cultured on culture media to which penicillin and streptomycin had been added: a pure culture of Histoplasma Capsulatum was obtained.

This experience is testimony of the value of cooperation between the clinician and the pathologist and the bacteriologist.

Dr. Moersch and his group have brought us a timely and stimulating report of excellent work.

Discussion

SEYMOUR M. FARBER, M.D., F.C.C.P.
San Francisco, California

Drs. Moersch, Weed and McDonald again have emphasized the value of bacteriologic studies of surgical specimens. Of particular interest to me were the three cases of coccidioidomycosis which illustrated how bacteriologic studies of a surgical specimen will alter a diagnosis. At the University of California we have recently reviewed 31 patients with coccidiodal infections. Seven of these patients entered with a provisional diagnosis of coccidoidomycosis. The remaining patients entered with varied diagnoses such as arthritis, chronic sinusitis, bronchitis, bone abscess, sarcoma of the ileum, malignancy of the skin, etc. By means of bacteriological studies a definite diagnosis of coccidiodal infection was established in 11 of these patients. Four of the others had biopsies but unfortunately cultures were not taken.

With the increasing number of cases of coccidioidomycosis being reported in various parts of the United States it is important that clinicians emphasize bacteriologic studies of surgical specimens. The six cases reported by the authors splendidly illustrate the need of these studies.

An Appraisal of the Results of a Six-Year Tuberculin and Mass X-Ray Survey of the Population of Asuncion: Based on More Than 100,000 Tests*

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Introduction

We can say that up to the last ten years the prophylaxis against contagious diseases was in the hands of Dispensaries whose task was reduced to the medical examination and treatment of all persons who came to the Dispensaries with likely symptoms of disease and very rarely as a preventive measure against disease. The only epidemiological basis on which prophylaxis rested was that of the above-mentioned means and compulsory reporting of disease. Under such conditions tuberculosis prophylaxis was well nigh illusory inasmuch as the only diseased persons examined were those who presented themselves at our only Dispensary. As for the compulsory declaration of disease the results obtained were very far below the real state of affairs in our case and it is also among other communities.

Diseased persons who were apparently healthy were never examined. Those suffering from contagious diseases (tuberculosis, syphilis, leprosy, etc.) entered every profession and took part in public activities although they were a constant source of danger as carriers of such diseases. It was absolutely essential to face the problem by examining everybody, whatever their occupation and station of life.

With the object of controlling the state of health of the general public and to take measures tending to improve and maintain it at a satisfactory level, a government decree, in October 1936, made the Health Card compulsory for all persons engaged in the manufacture and sale of food-stuffs and merchandise. In September 1938 this law was amplified to include all public servants, workmen, clerks, students, teachers, domestic servants, etc.

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*Report on the Prophylaxis and Epidemiology of the Tuberculosis Department, Asunción, Paraguay (Health Control Institute for Individuals and Communities, Director: Dr. A. R. Ginés, Profesor of Internal Pathology). Presented on the International Night Program, Thirteenth Annual Meeting, American College of Chest Physicians, Atlantic City, New Jersey, June 5, 1947.

Nevertheless, it was necessary to exercise special control over the chest diseases, which accounted for 50 per cent of the deaths in Asunción in 1941 (pulmonary tuberculosis, pneumopathies and heart diseases, respectively).

The Government assigned us the task of setting up the Organization and Administration of the Prophylaxis and Epidemiology Section of Tuberculosis in July 1941, and the results demonstrated the importance of the x-ray-tuberculin survey among the more important nuclei of the Capital (asylums, the Military School, Municipality, National Printing Office, textile factories, the interned and the staff of the reformatory for females, employees and attendants of the Clinical Hospital, and the relatives of persons suffering from tuberculosis).

Based on results obtained through our previous work the possibility of carrying out a mass x-ray and tuberculin survey in the city was favorably received and a decree issued in November 1941 widened the scope of the previous one thereby ordering the inclusion of the x-ray photograph as a compulsory prerequisite for securing the Health Card in Asunción.

The voluntary, private and collective medical examination was thereby complemented by the compulsory examination of all persons in active employment in our midst. As a matter of fact, voluntary examinations (which we still continue to perform without charge, and to which we give wide publicity on the radio and in the newspapers) have proved inadequate in obtaining good results. We are of the opinion that the same state of affairs will be found to exist in the majority of South American cities. As a consequence of this, and as an essential medium for getting satisfactory results, we recommend compulsory Health Cards which may be only issued after an x-ray and tuberculin examination has been made by a competent organization, such as our own Institute of Control of the Health of Persons and Communities, at least, in the case of populous cities.

It is understood that case finding can be conducted in two special ways which complement each other. One way is the "prophylaxis of the source" which is also called "centrifugal prophylaxis" the object of which is to discover new cases among those persons who are in contact with the patient. The second way is to carry out the preventive examination of all persons in the community without taking into account possible contacts. Mass x-ray examination of the community achieves this objective. It is by these means that we also carry out "centripetal prophylaxis" which discovers hitherto unknown sources of the disease among the apparently healthy. The periodical repetition of the examina-

tion will show lower figures for diseased persons owing to the constant control of the detected sources (See Fig. 1).

When we took charge in June 1941 of the organization and administration of Prophylaxis and Epidemiology of Tuberculosis in this country we adopted as a basis the above-mentioned method and, among other measures taken by us, such as the prophylaxis of the sources of the disease, sanitary education, B.C.G. vaccination, requests for the construction of sanatoria and hospitals, and the setting up of Social Insurance for people suffering from tuberculosis, we adopted in an ample fashion the system of the examination of communities in Asunción. In order to carry out the task before us we resorted to the compulsory Health Card system and the possession of which calls for periodic roentgen-photographic examination and tuberculin tests as well as other examinations whenever necessary.

By these means we have filed about 130,000 micra roentgen-photographs which correspond to 75-80,000 persons who were examined by us out of a total population of 126,000 according to the last census (1946).

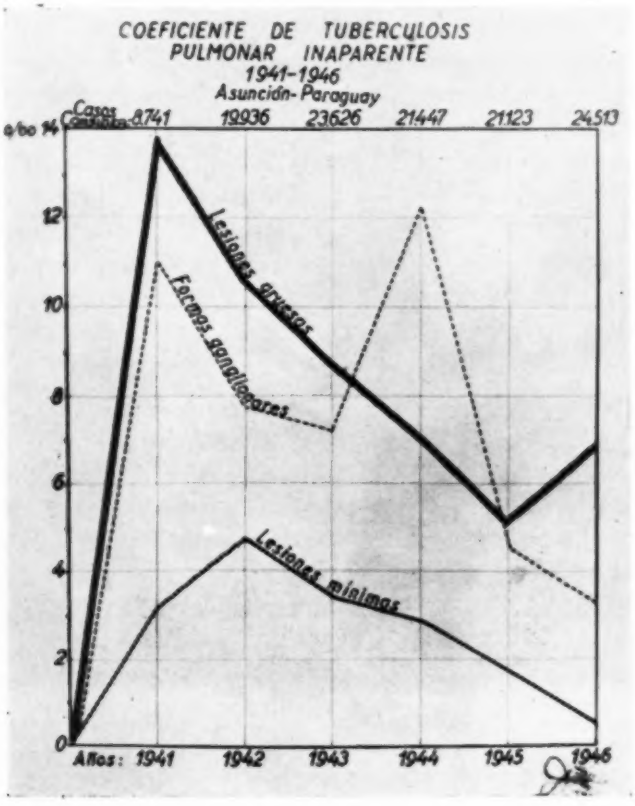

FIGURE 1

Diagnosis Technique

We include in our diagnostic technique the following:

a) Tuberculin test.

b) Abreu's roentgenphotograph or microfilm.

c) Standard x-ray picture (30 x 40 cm.).

d) Laboratory work including analysis of sputum, blood sedimentation rate and occasionally a haematological examination. Studies of the gastric contents are rarely carried out. Instead we have introduced bronchial washings (methods of Abreu and R. Fernandez).

a) *Tuberculin test:* In 1941 we carried out von Pirquet's test and from 1942 we used the intracutaneous test (Mantoux) with tuberculin solution 1/1000 as first test. Vollmer's patch was only used as an experiment. The second test, for non-reactors, was carried out directly with tuberculin 1/20. The tuberculin used in all these tests is that of synthetic medium (S.O.T.) made by the Instituto de Higiene de Montevideo.

b) *Abreu's X-ray photography:* Roentgenophotography is, without any doubt whatsoever, the axis on which the new style prophylaxis of tuberculosis revolves. While x-ray pictures would certainly be the ideal method, we are faced with the decided handicap of the price of the film which makes its use prohibitive in the examination of communities.

The film we use is Kodak Super XX which gives thirty-six exposures. The technique employed is as follows: 60 milliamperes and variable kilovoltage, according to the ventrodorsal diameter of the person examined, which runs from 50 to 80 Ky. The exposure time ranges from 5/10 to 9/10 of a second and very occasionally 1.5 seconds.

The rolls of film are then studied with the aid of a magnifying glass. It should be said that the diagnosis obtained by this method are almost never exact. They just serve as a means of selection. As a consequence all lung shadows, mediastinum or otherwise of the chest, must be checked first of all by means of an x-ray picture in order to reach an exact diagnostic conclusion as to the existence and the type of lesion discovered.

Potter's system, or rather roentgenphotography on 10 x 12 cm. films used in the United States, especially after its introduction in 1938 by Potter in collaboration with the General Electric X-ray Corp., has now been put into operation effective May 1947.

Examination Procedure

This is based mainly on the Health Card system, but we also examine people who solicit such an examination, and we carry

out examination of those persons sent to us by private practitioners or school doctors. In this way it may be said that we have examined practically everybody in every trade, whatever their sex or age.

The examination is the same in every case. Employees and professional workers are required to pay a fee of 3.00 pesos ($1.00) for Health Card while other persons are examined free of charge.

By methodical work we are able to take one x-ray photograph every minute, which gives us a total of 60 per hour. The procedure is best described as follows: (a) Presentation of the person to be examined in the Tax Office where he pays his fee. (b) Details and indexing. (c) Tuberculin test. (d) Micro-roentgenphotograph. The result of each examination is made known within 48 hours. In doubtful cases an x-ray photograph is requested and when negative reactors appear we use the second Mantoux test with 5 milligrams of tuberculin. If the x-ray picture examination confirms our suspicions we carry out examinations of the sputum and blood sedimentation rate. (See Plan).

The patient is then sent, along with the respective communication, to the Dispensary, or the Tuberculosis Hospital or to a private physician.

If the second tuberculin reaction is negative and the x-ray photograph is normal we carry out vaccination with B.C.G. (Rosenthal method).

On the Health Card which we issue to healthy persons we write down the number of examinations of the holder and the pertinent remarks relative to periodical examination, B.C.G. vaccination, etc.

The results of the different examinations are filed in special cabinets in numerical and alphabetical order, and also in books destined for special remarks on each type of examniation. The standard x-ray pictures are also filed in the same fashion.

Those persons found to be suffering from disease are reported to the Department of Hygiene every month. These monthly lists, along with the names sent in by other branches are filed in alphabetical order and also according to the individual's trade or occupation.

People with aortic and heart diseases are notified by means of their respective Health Cards to have a further and final clinical examination in a hospital or by a private physician.

We take an x-ray photograph (35 mm.) every two years of a normal person. The Health Card must be renewed every year and on these occasions we make a complete examination of the skin in order to detect syphilis and other contagious diseases.

The procedure of the community examination used in Asunción since the year 1941 is shown in the "Working Plan" Chart.

Working Plan of the Health Control Institute for Individuals and Communities

VOLUNTARY EXAMINATION (free of charge)	COMPULSORY EXAMINATION
TAX OFFICE Tax: Gs. 3.00 ($1.00)	
CARD RECORD TUBERCULIN TEST X-RAY PHOTOGRAPH	RENEWAL of HEALTH CARD CHECK ON PREVIOUS YEAR'S HEALTH CARD SKIN EXAMINATION
FINAL RESULTS (48 hours later) Normal HEALTH CARD or TICKET (free Health Card)	PENDING RESULTS of OTHER EXAMINATIONS Standard Tuberculin Test. Standard X-Ray Picture. Cardiovascular control. Laboratory examination.
FINAL RESULTS	
Normal Non-reactor B.C.G. Vaccination HEALTH CARD or TICKET (free Health Card)	Patient HOSPITALS DISPENSARIES PRIVATE PHYSICIANS SOCIAL INSURANCE

Epidemiological Data

Before we summarize the results obtained it should be mentioned that in Asunción the actual tuberculosis mortality is 20 per 10,000. The infection index as revealed by tuberculin tests varies from 91 to 97 per cent for the population taken as a whole and shows very high figures for the first years of life: 50-60 per cent from 0 to 5 years; 70-80 per cent from 6 to 10 years; and 80-90 per cent at the age of 15. Latent morbidity was 2-3 per cent during 1941 and 1942 and showed 6-20 per cent chest lymph node tuberculosis in children under the age of six and 4-10 per cent for those 6 to 10 years of age.

These figures demonstrate the period of massive tuberculosis through which we are passing. The epidemic curve, as judged by the tuberculosis mortality index compiled since 1895, must have commenced during the last century since very high percentages, almost comparable to those of the present, have been registered since the beginning of the present century. The lack of statistical data does not permit further information.

Results

Owing to the extent which a full description would demand we shall limit ourselves to a summary of the full report presented before the Seventh Pan-American Congress of Tuberculosis (Lima, Perú, January 1947).

The results and conclusions arising from the study of 105,898 persons who underwent the tuberculin tests (repeated on non-reactors up to 5 milligrams Mantoux), 119,386 x-ray photographs, 5,000 roentgenograms of different chests (30 x 40 cm.) and some laboratory work on sputum and blood sedimentation rate, are complemented by the prognostic study of the different kinds of lesions found during our investigations, and also that of apparently normal people without radiological evidence of pulmonary lesions who some time after becom ill or die from tuberculosis.

A) *Tuberculin and roentgenphotography test results* can be divided in a) Epidemiological and b) Clinical.

a) *Epidemiological contribution:*

1) The high annual positive tuberculin index (91-97 per cent) for all ages, 50-60 per cent for the first five years of life; 60-80 per cent for children 6-10 years of age and as high as 95 per cent around 20 years of age; the high index of latent morbidity appraised for the lymph node form is 6-20 per cent for the first five years of life, 4-11 per cent from 6-10 years of age, and 1-2 per cent up to the age of 15, to which must be added the minimal lesions in a proportion of rather less than 1 per cent, and gross lesions whose proportion varies between 1-2 per cent from 16 to 50 years of age and which is subject to a considerable increase after the age of fifty which brings about a percentage of 3-4, go to show that the city is going through a period of massive infection, which is in accord with its high ratio of tuberculosis mortality (about 20 in 10,000).

Among the *lymph node* forms we include hilar, mediastinal and tracheobronchial lymph nodes, some of them with infiltrative pulmonary lesions all of which can be styled as "infantile forms" or "primary infection." Among the *minimal lesions* we include nodules, local thickness of the broncho-vascular tissue with exudative aspects, fuzzy at the edges, excluding the hard tissues and nodules. *Gross lesions* include all those forms known as "reinfection" whatever their type (exudative, proliferative, mixed or destructive) and whatever the extent, be it infiltrative or phthisic, in other words, a large part of the minimal and all the moderately advanced and far advanced forms according to the North American

classification. The active pleural forms are classified in this group while the residual lesions are excluded.

2) The mass x-ray examination, or the search for tuberculosis among the apparently healthy, has brought about a progressive annual decline in the infection index (including the hyperergics) and of the latent morbidity index in its adenopathic forms, both minimal and gross. These reductions, which correspond to all ages, although more pronounced among those of tender age, taken as a whole show the following figures for 1941, 1942, 1943, 1944, 1945 and 1946 respectively (See Fig. 2).

b) *Clinical contribution:* A clinic-radiological appraisal of the different tuberculin allergy based on 2,000 reactors in each group reveals:

1) The negative reaction allows us practically to exclude latent active tuberculosis. We have to make an exception of healthy normergics (nonreactors) under the age of 20 in whom we have found 0.15 per cent of tracheobronchial lymph nodes and 0.50 per cent of hilar lymph nodes of supposed bacillary etiology which would be found during the so-called pre-allergic period; and 0.10 per cent of infiltrative processes whose bacillary etiology could not be determined. The presence of calcifications in persons from

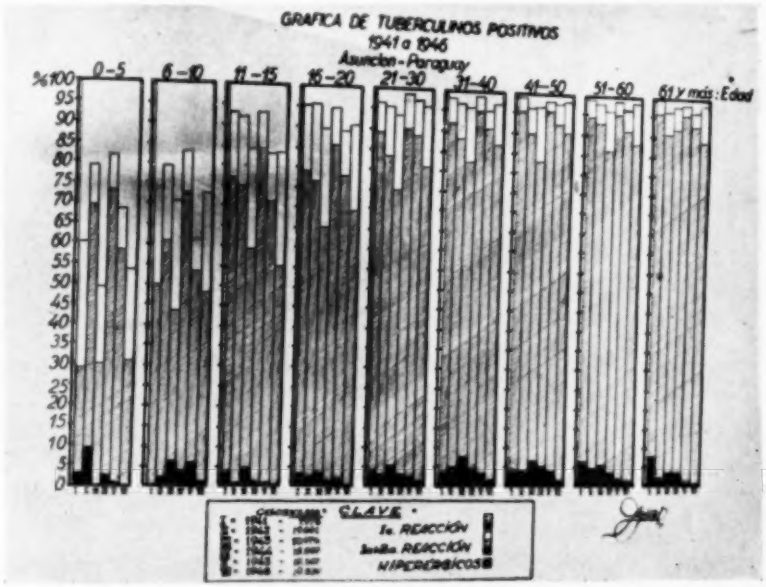

FIGURE 2

Allergic curve: 96.4; 95.8; 91.3; 96.9; 93.8; and 93.9 per cent.
Hyperergic curve: 4.2; 3.5; 5.3; 3.4; 2.1; and 1.1 per cent.
Adenopathic forms: 1.11; 0.78; 0.72; 1.21; 0.44; and 0.37 per cent.
Minimal lesions: 0.32; 0.48; 0.34; 0.29; 0.17; and 0.05 per cent.
Gross lesions: 1.38; 0.06; 0.87; 0.70; 0.51; and 0.68 per cent.

2 to 60 years of age in this group to the extent of 9 per cent demonstrates the reality of the "extinction of the allergy," since such calcifications do not appear to have any relation with the coccidioidine histoplasmine allergy recently tested by us, while they do respond to B.C.G. with shortening of the pre-allergic period, which seems to indicate that we should classify them among the latent allergies, or hidden allergies according to Willis-Sayé.

2) The reactors with one or two plus reaction (5 to 9 mm. and 10 to 19 mm.) as well as the reactors to the second test, and the so-called doubtful reactors (less than 5 mm.) reveal 2-3 per cent of lung or lymph node lesions; while those with 3 or 4 plus reaction (more than 19 mm. or a phlyctena) show 8.10 per cent of the same type of lesions.

B) *Radiographic Results:* Among 5,000 radiographies which were taken as a result of suspicions arising from the microfilm, 29 per cent corresponded to lesions originating in tuberculosis, 34.44 per cent to residual forms and 32.53 per cent to cardioaortic lesions or other costal, lung or mediastinal abnormalities of different kinds. For every three microfilms one corresponded to tuberculous lesions, one to sequelae and the remainder to the anomalies mentioned.

Among the different clinico-radiological classifications of tuberculosis we have preferred to use that of the Spaniard Diez Fernandez because of its greater suitability to our needs. We have effected some slight modifications, without altering it in its essence, in order to try to simplify its application. For the sake of brevity we shall only present the lesions in their corresponding principal groups:

I-A—lymph node forms, 35.14 per cent.
I-C—disseminating forms, 9.92 per cent.
III-A—infiltrative forms, 26.8 per cent.
AB—phthisic forms, 28.11 per cent.

If we consider that the first two groups cited correspond to children and young people, we find that 45 per cent correspond to infantile form classified as "primary infection," while 55 per cent correspond to adult phthisic forms or "reinfection."

Among the so-called primary infection the hilar lymph nodes were present to the extent of 54.32 per cent, the tracheobronchial lymph nodes 33.96 per cent and enlargements of the lymph nodes accompanied by infiltrative lesions, 10.86 per cent. Among the so-called haematogenous dissemination 5 per cent correspond to miliary forms, 60 per cent to partial dissemination in one or both lungs, and 35 per cent to pleural forms.

Among the infiltrative forms, which are considered as the be-

ginning of tuberculosis in adults, 28 per cent are cases with cavity formation at the time of the first examination and 72 per cent are still free of cavity formations. Among the phthisis forms the caseous exudatives and their lobar and lobular forms account for 5.63 per cent, proliferatives 57.92 per cent, the destructives 11 per cent and the fibrocaseous 25.23 per cent.

As can be seen any one of the anatomoclinical types of tuberculosis can appear without clinical symptoms and become a radiological discovery in the "apparently healthy."

Prophylaxis by means of the mass x-ray examination is thereby carried out in all the pulmonary forms of tuberculosis and, with the exception of the phthisic forms, in which the possibilities of cure are very remote, as well as the fatal disseminating forms, we can expect genuine success in 81 per cent of the cases of discovered lesions, in which are included the lymph node, infiltrative and partial disseminating forms.

C) *Laboratory work in the diagnosis of latent lung tuberculosis:*

The direct examination of the sputum and the blood sedimentation rate is referred to exclusively.

a) *Acid-fast bacilli and expectoration in relation to lung lesions:*

III Am *(Infiltrative Lesions:*	Cases	Percentage
With sputum: with bacilli	5	16.6
(48 per cent) Without bacilli	25	
Without sputum	34	
III An *(Infiltrative cavity forms):*		
With sputum: with bacilli	27	45.0
(83 per cent) Without bacilli	32	
Without sputum	12	
III Bo *(Caseous exudative phthisis):*		
With sputum: with bacilli	4	66.6
(66 per cent) Without bacilli	2	
Without sputum	3	
III Bp *(Proliferative phthisis):*		
With sputum: with bacilli	10	22.0
(61 per cent) Without bacilli	35	
Without sputum	29	
III Bq *(Destructive phthisis):*		
With sputum: with bacilli	8	66.0
(93 per cent) Without bacilli	4	
Without sputum	1	
III Brl *(Common fibrocaseous phthisis):*		
With sputum: with bacilli	31	69.0
(81 per cent) Without bacilli	14	
Without sputum	6	

Sputum is present in 83-93 per cent of the cavity formations and in a half or perhaps rather more than a half of the infiltrative and phthisic forms of the disease. The direct examination of sputum shows a positivity of 16.6 per cent in the simple infiltrative forms, 45 per cent in those forms with cavities, 22 per cent in the proliferative phthisis, 66.6 per cent in broncho-pneumonic phthisis, 66 per cent in the destructive forms and 69 per cent in the fibro-caseous lesions.

b) *Blood sedimentation rate in relation to lung lesions:*

III Am *(Infiltrative Lesions):*	Cases	Percentage
Up to 10 mm.	24	36
11-30 mm.	31	47
31-70 mm.	10	15
More than 70 mm.	1	1.5
TOTAL	166	

III An *(Infiltratives with cavity formations):*		
Less than 10 mm.	6	16
11-30 mm.	10	26.6
31-70 mm.	17	44.5
More than 70 mm.	5	13.3
TOTAL	38	

III Bo *(Exudative caseous phthisis):*		
Less than 10 mm.	2	22.2
11-30 mm.	3	33.3
31-70 mm.	1	11
More than 70 mm.	3	33.3
TOTAL	9	

III Bp *(Proliferative phthisis):*		
Less than 10 mm.	21	33
11-30 mm.	13	36.5
31-70 mm.	18	29
More than 70 mm.	1	1.74
TOTAL	53	

III Bq *(Destructive phthisis):*		
Less than 10 mm.	1	11
11-30 mm.	2	22.22
31-70 mm.	3	33.33
More than 70 mm.	3	33.33
TOTAL	9	

III Brl *(Common fibrocaseous phthisis)*:	Cases	Percentage
Less than 10 mm.	11	28
11-30 mm.	8	21
31-70 mm.	9	23
More than 70 mm.	10	26
TOTAL	38	

As can be seen the blood sedimentation rate appears as normal in 36 per cent of the infiltrative and proliferative forms, 28 per cent in the fibrocaseous lesions, 16 per cent in the infiltratives with cavity formations and 11 per cent in the destructive forms. Although without diagnostic value this goes to show the stage of toxic repercussion of the disease in the organism.

D) *The contribution of successive surveys to the Pathogenesis of tuberculosis.*

Having taken out of our files at random 8,113 index cards of persons of both sexes whose ages ranged from 2-60 years who had been examined at intervals of one or two years, most of them twice, some three times and, very exceptionally, four times, we made a brief study of the tuberculin reactions and the appearance of lesions. This served to demonstrate the following:

Allergic fluctuation is a common occurrence. Allergy appears in 80 per cent of nonreactors within the term already mentioned. Allergy, also, attenuates, dies out or exacerbates itself. The loss of allergy is an undeniable fact and its frequency was found to be 0.28 per cent among hyperergics (+++ and ++++), 2.4 per cent among allergics with two plus reaction, 5.1 per cent among those with one plus reaction and 9 per cent among hypoergics. This gives the impression that the aforesaid loss comes about gradually.

Nonreactors show lesions without the reaction having varied to the extent of 1.33 per cent which are limited to a large amount of the lymph nodes (pre-allergic period); when a positive change occurs of this group show lesions (primary lesions?), distributed among enlarged lymph nodes (93 per cent) and pulmonary infiltrations (7 per cent).

Lesions which appear among allergics with or without change in their respective tuberculin tests show a frequency of 1.06, 1.29, 1.09 and 3.40 per cent among hypoergics and allergics with 1, 2 or 3 plus reaction respectively. Enlargement of the lymph nodes predominate overwhelmingly over the infiltrative, disseminating, pleuritic and phthisic forms as the first radiographic manifestation of the disease.

It does not seem to be the primary complex nor the pleural

reaction but the adenopathy which is the first detectable lesion before and during the development of the allergy. This leads us to think that the "complexes" are in reality advanced radiological stages of the lesion. Nor can discrimination be made in the adult between first infection and reinfection upon the basis of the anatomo-radiological lesions even when prior results of tuberculin tests of the individual had been known.

Treatment

The treatment of the latent lesions could not be appraised among us because of the only recent inauguration of a well-equipped Tuberculosis Sanatorium in our midst. Good preventive results were obtained among normergics with B.C.G. vaccination.

Prognosis

Prognosis of latent pulmonary tuberculosis is presented from the point of view of the spontaneous evolution of the lesions (without active collapse therapy or sanatorium treatment) and of its "quoad vitam" significance.

a) *The lymph node enlargements* recede in 84.2 per cent, remain stationary in 12.8 per cent and evolve, presenting infiltrative (1.4 per cent) and phthisic forms (1.4 per cent). *Minimal lesions* disappear to the extent of 40 per cent, remain stationary in 20 per cent and evolve in phthisic forms in 40 per cent. *Infiltrative forms* heal in 43 per cent, recede partially, leaving some nodules, in 1.5 per cent, and show pleural complications in 3.1 per cent, remain stationary in 43.7 per cent and evolve in phthisic forms in 7.7 per cent of the cases. Phthisic forms do not heal, but evolve in 92.9 per cent and recede partially in 7.1 per cent.

b) *"Quoad vitam" prognosis:* In order to study this we have consulted the tuberculosis mortality lists of the past six years in order to identify those deceased persons who figured in our register. Moreover, we have had recourse to our files in order to find the names of all those persons who died from tuberculosis during 1943-46.

In spite of possible errors accruing from the use of this technique we believe it to be of interest to publish the results obtained.

It can be said that the lymph node forms without concomitant lung lesion show a 100 per cent favorable life prognosis. Among the other forms, fibrocaseations show a high mortality figure (17 per cent) and are followed by caseous pneumonic phthisis (12.17 per cent), disseminating cases (4.24 per cent), infiltrations with cavity (5.25 per cent), infiltrative and productive cases (4.4 per cent), and the destructive cases (5.3 per cent). Deaths generally occur within two or three years or longer but rarely in the fourth

year whatever the form. Eighty per cent of the deaths occur within the first two years.

We desire to call attention to the fact that the prognosis for certain healthy allergics in 1, 2 or 3 years is no more favorable than that corresponding to the discovered lesions. These cases account for about 60 per cent of the total of deaths from tuberculosis in our midst. They form a part of what Flatzek preferred to style "additional mortality" and as far as our case is concerned we share his opinion, by which we mean to say that they are a result of precarious means of livelihood and poverty.

GENERAL CONCLUSIONS

1) Tuberculosis case-finding among apparently healthy individuals should be carried out systematically by means of: (a) community examination; (b) examination of the family and known contacts of tuberculous persons and (c) examination of admission cases to the general hospitals.

2) In large cities the Community Examination should be carried out at fixed places which the general public can reach easily, while mobile x-ray equipment can be utilized to attend country people and the Army.

3) Photofluorography (35 mm.) is the most economic and efficient method of discovery of latent, endothoracic lesions. For this reason alone, its originator, Manuel de Abreu, should have his place among the benefactors of humanity.

4) Tuberculin reaction will demonstrate the existence of tuberculous infection and its use should be the rule in the clinical and epidemiological study of tuberculosis.

5) The standard x-ray picture will decide in every case whether tuberculous lesions which had been suspected from the roentgenphotograph really exist; but only the clinic and the complementary elements of the laboratory can decide definitely what is the diagnosis and its importance.

6) The Community Examination should not stop at the discovery of new sources of the disease, but should contribute towards its control and treatment by making the cases known and sending them to specialists in phthisis or a tuberculosis center.

7) Since the roentgenphotographic-tuberculin method is practically infallible in the examination of healthy normergics, the subsequent use of B.C.G. vaccine is extremely valuable as a final prophylaxis measure.

8) The Card Index File and the Register of Results should merit special attention. They serve as sources of statistical information and of investigation and also cut out a great deal of routine work

TUBERCULIN TESTS — 105,858 Cases:

Age	0-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	40	56	72	124	262	311	685	612	1525	1097	1088	555	614	264	375	155	110	31	4771	3205	7976
1942	32	32	79	144	914	712	2062	1901	4663	2693	3140	1089	1006	288	570	134	169	33	12635	7026	19661
1943	56	56	288	387	1546	1196	2416	1787	3746	2262	2572	1097	1403	619	885	283	300	77	13212	7764	20976
1944	66	70	100	160	1179	825	2137	1757	3795	2267	2371	1084	1216	509	762	233	281	87	11907	6992	18899
1945	31	29	56	107	1057	764	2186	1776	3631	2032	2509	926	1440	501	883	201	307	71	12100	6407	18507
1946	55	70	209	256	1105	910	2355	1601	4118	2042	2670	1012	1550	554	793	179	300	60	13155	6684	19839

PERCENTAGE TOTAL OF REACTORS — According to age and sex:

Age	0-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	57.5	62.3	80.0	71.7	94.3	92.4	94.4	96.1	98.1	97.1	99.1	98.0	99.5	97.7	99.2	99.1	99.6	100.0	97.3	95.0	96.4
1942	78.0	81.2	78.4	80.4	93.8	90.0	95.5	95.0	97.3	95.1	97.4	97.1	96.1	96.5	98.5	99.1	95.1	100.0	96.5	94.2	95.8
1943	53.5	44.6	67.3	72.0	85.8	81.3	89.3	87.2	93.1	91.1	97.0	95.4	96.5	95.6	96.7	95.6	98.3	98.6	91.5	88.7	91.3
1944	86.3	78.5	84.0	85.0	94.2	93.2	96.2	93.3	97.1	96.3	98.6	97.5	98.9	97.1	98.6	98.1	98.3	97.9	97.6	95.6	96.9
1945	61.2	75.8	67.5	59.4	87.7	79.0	91.6	86.5	96.6	94.0	97.0	96.0	98.0	97.0	97.0	97.5	95.0	98.0	94.5	90.5	93.0
1946	61.8	47.0	73.0	72.9	85.3	81.6	93.6	88.3	97.4	92.5	97.8	96.2	98.6	97.0	98.5	95.4	99.0	98.2	95.4	89.9	93.9

PERCENTAGE REACTORS TO 0.1 MILLIGRAM S.O.T. (first mantoux):

Age	0-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	25.0	32.0	58.0	45.9	79.5	74.8	80.5	77.8	92.1	84.9	95.3	87.5	96.5	93.7	95.2	92.1	96.0	93.7	90.3	81.6	86.9
1942	68.7	68.7	60.7	61.7	78.5	71.4	78.7	76.0	84.6	81.2	89.4	86.6	90.3	88.8	93.0	93.9	89.2	97.0	85.0	79.9	83.1
1943	35.7	25.0	42.0	43.3	61.6	56.3	65.7	63.8	76.8	71.1	84.1	79.7	85.1	78.0	87.0	83.2	88.0	85.6	75.0	67.9	72.7
1944	75.7	71.4	75.0	73.0	84.7	83.1	87.6	82.7	91.0	89.3	94.9	94.0	95.8	93.8	94.7	94.7	94.4	95.7	91.6	88.3	90.4
1945	48.3	68.9	51.5	47.4	76.7	64.0	80.6	75.5	90.0	85.0	93.0	90.0	93.0	90.0	93.0	92.5	92.0	95.0	87.5	81.0	85.0
1946	40.0	24.2	44.0	41.3	53.7	55.8	72.5	65.2	85.0	74.2	89.5	82.8	91.6	85.1	89.0	84.9	90.0	83.2	81.3	70.5	78.0

PERCENTAGE HYPERERGIC REACTORS (3 and 4 plus reactors):

Age	0-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	5.0	1.7	..	..	4.5	3.8	3.0	4.3	4.7	3.7	4.3	3.5	4.7	3.7	7.2	3.8	8.0	6.4	4.6	3.6	4.2
1942	9.3	9.3	2.5	2.0	1.5	2.1	2.0	2.6	2.8	4.7	4.8	5.6	4.6	3.4	4.0	6.6	1.7	3.0	3.2	3.9	3.5
1943	..	..	4.0	6.9	3.9	4.8	3.0	4.8	4.9	6.1	7.1	8.7	6.3	7.0	5.6	4.5	4.0	2.6	5.0	5.9	5.3
1944	3.0	1.4	4.0	5.0	1.7	2.1	1.6	2.7	3.0	4.3	3.9	6.0	4.8	6.8	3.7	2.5	2.4	5.7	3.0	4.1	3.4
1945	3.2	..	3.5	8.4	1.2	1.1	1.6	2.4	2.1	3.4	3.4	5.2	4.3	4.2	3.0	2.5	1.3	1.4	2.5	3.2	2.1
1946	..	..	1.0	1.9	0.8	0.6	0.3	0.9	0.7	1.3	1.7	1.8	1.1	2.1	1.4	1.1	..	1.6	1.0	1.2	1.6

ABREU ROENTGENPHOTOGRAPHS—Total of Cases, 119,386:

Age	0-2		3-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	24	39	38	47	182	330	302	707	708	809	1454	1131	1008	547	554	258	341	130	97	35	4708	4033	8741
1942	14	11	21	24	83	137	859	671	2003	1696	4200	2497	2919	1031	1604	528	997	216	367	58	13067	6869	19936
1943	15	18	52	45	351	450	1658	1232	2607	1975	4258	2619	2963	1236	1646	663	1047	324	369	98	14966	8660	23626
1944	29	25	61	70	125	194	1324	973	2557	1919	4267	2493	2677	1196	1347	598	883	264	344	101	13614	7833	21447
1945	7	8	32	38	92	133	1216	901	2385	2000	4091	2399	2881	1064	1643	548	1012	240	349	84	13708	7415	21123
1946	21	15	91	81	294	373	1422	1055	2873	1807	4956	2649	3208	1289	1894	674	1028	259	425	99	16212	8301	24513

PERCENTAGE LYMPH NODES AND CHILDHOOD TYPES OF TUBERCULOSIS:

Age	0-2		3-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	12.5	12.8	10.5	8.5	3.8	4.2	1.0	1.4	1.5	1.3	0.8	0.4	0.2	0.9	..	..	0.3	..	..	..	0.91	1.34	1.11
1942	35.7	45.5	14.3	20.8	9.7	11.7	1.6	2.5	0.7	0.5	0.5	0.4	0.4	0.5	0.2	..	0.4	0.9	..	..	0.63	0.97	0.78
1943	20.0	6.2	9.6	15.9	4.6	3.7	1.8	1.3	1.0	0.3	0.4	0.3	0.2	0.2	0.06	0.1	..	..	..	..	0.72	0.71	0.72
1944	3.4	..	10.0	5.7	8.8	3.1	1.8	2.0	1.4	0.9	1.1	0.9	0.9	0.6	0.9	1.3	0.6	0.4	..	.	1.26	1.13	1.21
1945	14.3	12.5	..	5.2	7.6	5.2	10.0	0.8	0.3	0.4	0.4	0.2	0.1	0.6	0.2	..	0.1	0.4	0.2	..	0.40	0.52	0.44
1946	38.0	13.3	8.7	8.6	4.7	4.0	0.7	0.8	0.1	0.2	0.02	0.07	..	..	..	..	..	..	.	..	0.27	0.49	0.37

PERCENTAGE OF MINIMAL LESIONS:

Age	0-2		3-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	..	..	..	..	0.5	..	0.3	..	0.5	0.5	0.3	0.1	0.5	..	0.7	0.4	..	0.7	..	..	0.42	0.19	0.32
1942	..	..	..	..	..	..	0.3	..	0.3	0.3	0.8	0.3	0.3	0.5	0.5	0.2	0.9	0.4	0.5	..	0.58	0.27	0.48
1943	..	..	1.9	..	..	..	0.3	0.3	0.5	0.3	0.4	0.2	0.4	0.3	0.2	..	0.5	0.6	..	..	0.39	0.25	0.34
1944	..	4.0	..	..	..	..	0.07	0.1	0.2	0.05	0.3	0.2	0.3	0.6	0.3	0.6	0.3	..	2.0	1.0	0.31	0.25	0.29
1945	..	..	..	..	..	..	0.08	0.1	0.1	0.1	0.1	0.2	0.2	0.1	0.1	0.5	0.4	0.4	0.3	..	0.17	0.18	0.17
1946	..	..	..	..	0.6	0.2	0.07	0.1	0.03	0.05	..	0.03	0.1	..	..	0.1	..	..	0.2	..	0.04	0.06	0.05

PERCENTAGE OF GROSS LESIONS:

Age	0-2		3-5		6-10		11-15		16-20		21-30		31-40		41-50		51-60		Over 61		Total	Total	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	Male	Female	Total
1941	..	..	..	..	1.1	0.3	0.7	..	0.5	1.1	1.6	1.7	2.1	1.8	2.5	0.4	2.0	1.5	4.1	2.8	1.66	1.06	1.38
1942	..	9.1	..	..	1.2	..	0.3	0.1	0.3	0.4	1.1	0.7	1.2	1.2	1.9	1.1	2.1	2.7	3.7	1.7	1.20	0.77	1.06
1943	..	..	..	..	0.3	..	0.2	0.3	0.6	0.3	0.8	1.0	0.8	0.6	1.1	1.1	2.5	2.1	3.5	4.0	0.93	0.76	0.87
1944	..	..	..	..	..	..	0.1	0.1	0.2	0.1	0.6	0.7	1.2	0.6	1.5	0.6	1.7	1.1	3.2	1.0	0.84	0.46	0.70
1945	..	..	..	..	..	..	0.1	0.3	0.2	0.1	0.4	0.7	0.4	0.5	0.9	1.1	1.1	..	0.1	3.8	0.51	0.51	0.51
1946	..	6.6	..	..	..	0.5	0.07	0.3	0.3	0.5	0.4	0.4	0.8	0.7	0.9	1.3	2.0	1.1	2.6	3.0	0.68	0.62	0.68

in future examinations of the same patient as valuable time and films are economized.

9) In order that the final object of the work being done and its appraisal can be further improved, connections should be maintained with other centers of prophylaxis, treatment and investigation of the disease.

10) Although Sanitary Education should not be neglected, the Compulsory Health Card should be resorted to in order to ensure a rapid, uniform and comparative survey of the prophylaxis and epidemiology of tuberculosis in large cities.

11) The Community Examination plays a manifest and rapid part in the decline of infection and tendency to tuberculosis. Its labor is backed up by B.C.G. vaccination and modernization in the form of sufficient beds in the sanatoria destined for the purpose, and by the establishment of Social Insurance. In spite of this, however, additional tuberculosis mortality, which accounts for 60 per cent of the total tuberculosis mortality among us, can only be checked by means of economic and social measures which regulate and guarantee the most elementary needs of the people.

CONCLUSIONES GENERALES

1) El descubrimiento de casos de tuberculosis entre personas aparentemente sanas debe ser llevado a cabo sistemáticamente por medio de: (a) examen de la colectividad; (b) examen de la familia y de los contactos conocidos de personas tuberculosas y (c) examen de los casos admitidos a los hospitales generales.

2) En ciudades grandes el Examen de la Colectividad debe llevarse a cabo en lugares fijos, a los que el público en general puede acudir fácilmente, mientras que el equipo radiográfico móvil puede ser utilizado para servir a la gente del campo y al Ejército.

3) La roentgenofotografía (35 mm.) es el método más económico y eficaz de descubrir lesiones endotorácias latentes. Basta esta razón para que su inventor, Manuel de Abreu, ocupe un lugar entre los benefactores de la humanidad.

4) La reacción a la tuberculina demuestra la existencia de la infección tuberculosa y debe emplearse siempre en el estudio clínico y epidemiológico de la tuberculosis.

5) La película radiográfica regular decidirá en cada caso si verdaderamente existen lesiones tuberculosas que se habían sospechado en la roentgenofotografía; pero sólo los hallazgos clínicos y los exámenes complementarios de laboratorio pueden decidir definitivamente cuál es el diagnóstico y qué importancia tiene.

6) El Examen de la Colectividad no debe terminar con el descubrimiento de nuevas fuentes de la enfermedad, sino que debe contribuir a su control y tratamiento. Con este objeto se deben

dar a conocer los casos y se debe mandarlos a especialistas en tisis o a centros para tuberculosos.

7) Ya que el método roentgenofotográfico-tuberculínico es virtualmente infalible en el examen de los normérgicos sanos, el empleo subsiguiente de la vacuna B.C.G. es sumamente valioso como medida profiláctica final.

8) El Archivo de Tarjetas de Indice y el Registro de Resultados deben merecer atención especial pues sirven como fuentes de información estadística y de investigación y eliminan, también, mucho trabajo rutinario en exámenes futuros del mismo paciente, economizando así valioso tiempo y películas.

9) Para alcanzar el objeto final de la labor que se lleva a cabo y para su mejor valuación, deben mantenerse relaciones con otros centros de profilaxia, tratamiento e investigación de la enfermedad.

10) Aunque no se debe descuidar la Educación Sanitaria, se debe recurrir a la Tarjeta de Salud Obligatoria a fin de asegurar un censo rápido, uniforme y comparado de la profilaxia y epidemiología de la tuberculosis en las grandes ciudades.

11) El Examen de la Colectividad desempeña un papel claro y rápido en la disminución de la infección y la tendencia a la tuberculosis. Respaldan su labor la vacunación con B.C.G., la modernización, en la forma de suficientes camas en los sanatorios destinados para este propósito, y el establecimiento del Seguro Social. Sin embargo, a pesar de esto, la mortalidad por la tuberculosis, que entre nosotros es responsable por el 60 por ciento de la mortalidad total, sólo puede ser combatida por medio de medidas económicas y sociales que regulen y garanticen las necesidades más elementales de nuestra gente.

Bagasse Disease of the Lungs*

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The product remaining after extraction of sugar from sugar cane is called *bagasse*. This material is frequently burned, either as fuel or for disposition, but in some areas is baled for future conversion, by processing, into insulating and acoustic board. It may remain in the field for some months before use.

In 1941 Jamison and Hopkins[1] reported pulmonary disease presumably resulting from inhalation of bagasse dust. In 1942 Castleden and Hamilton-Paterson[2] and Gillison and Taylor[3] reported six additional cases. In 1943, Sodeman and Pullen[4] reported a case with needle biopsy of the lung and described a section of the lung of another patient who had died of the disease. In 1944 the same authors[5] summarized the findings in eleven cases, including the biopsied case reported in 1943. They discussed the possible etiologic factors underlying the pulmonary picture, and, in a separate report with Pinto and Pearson,[6] demonstrated the relative unimportance of the silica fraction. In the same year Jamison, Bryan and Day[7] reported an additional case, bringing the total number on record to twenty. The present report concerns the findings in four additional patients.

All cases previously reported have occurred in men. One of the present group is that of a colored female, thirty-four years of age, who gave a history of long continued exposure to dust arising from insulating board *after* processing. Her history does, however, include exposure to the dust of bagasse before processing. In none of the other cases cited has the pulmonary picture been noted following exposure to dust after processing, and, since this patient had been exposed to both types of dust, a claim of disease appearing after exposure to processed dust cannot be made.

The outstanding clinical findings are summarized in Table 1.

As in the group previously reported,[5] sex and age depend on selection of workers in bagasse. Only one case (Case No. 2) was a female, and the ages varied from twenty-six to fifty-seven. All were Negro. Length of exposure before the development of symptoms varied from four to seventy-eight weeks, but in three of the

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SUMMARY OF CLINICAL DATA ON CASES REPORTED

Case No.	Age	Race	Length of Exposure (Weeks)	Cough	Dyspnea	Sputum	Hemoptysis	Night Sweats	Chills and Fever	Loss of Weight (Pounds)	Pharyngeal Irritation	Duration of Symptoms before Admission (Weeks)	Cyanosis	Examination of Chest	Roentgenologic Picture of Chest	Days of Hospitalization	Follow-up
1	57	C	10	+	+	+	−	+	+	30	+	6	+	Rales over both bases posteriorly	A mottled infiltration of both lung fields spreading from the hilum to the periphery	3	Death with autopsy
2	34	C	78	+	+	+	−	+	−	15	+	8	−	Vocal fremitus increased with B-V breathing right lung posteriorly	As above	7	Complete clearing
3	26	C	4	+	+	+	−	−	−	+ Amt.?	+	3	−	Fine rales scattered throughout both lung fields	As above	2	Complete clearing
4	42	C	8	+	+	+	+	+	+	+ Amt.?	−	4	−	Fine rales throughout both lung fields	As above	9	Lungs almost clear to roentgen-ray in two months

four instances fell in the usual range of three weeks to two months.

Symptomatically, cough, dyspnea, and production of sputum, were outstanding findings. Dyspnea appeared suddenly, as a rule, and became sufficiently severe to force the patient to bed. Hemoptysis occurred but once in this group and generally is uncommon. The sputum was scant and mucoid with a whitish appearance. Fever, at times with chills, was found in two patients. In general the fever curves and respiratory and cardiac rates followed the trend described in the previously reported series of eleven cases.[5]

Examination of the chest revealed findings which were not striking, and which were meagre in proportion to the roentgenologic picture. Scattered and patchy rales with minor changes in breath and voice sounds were heard. The roentgenologic findings were those of a bilateral mottling more dense in the hilar regions. Invariably clearing of the process was found in the follow-up examinations.

Previous studies have disclosed[5] that tubercle bacilli are not found in the sputum, even with concentration methods. Fungi have not been found consistently, and bronchoscopy has shown little. The sputum has shown, in general, a cellular content of both polymorphonuclear leukocytes and lymphocytes. Eosinophiles have been absent. In the blood, moderate leukocytosis is the common finding, with slight shifts to the left. Sedimentation rates are increased and, later on in the clinical picture, mild increases in the hematocrit and red blood count have been observed. Blood cultures, agglutination reactions for typhoid, paratyphoid, tularemia, and undulant fever have been negative. The present four cases conform to these findings. The Mantoux test has not been consistent. In the four cases now reported, two were negative, one positive, and in one case was not done.

Treatment has consisted primarily of bed rest during the acute stage of the disease together with supportive and palliative measures.

The clinical picture, as described above, represents the major findings as seen in our institution. It should be emphasized that only those patients have been seen who have sufficiently severe complaints to seek hospitalization. It is likely that a more mild clinical picture occurs, and, possibly, roentgenologic evidence of pulmonary involvement without symptoms at all. Surveys of workmen in bagasse would be necessary to establish these findings and the author has not had the opportunity to carry out such a survey.

The cause of the clinical picture is unknown. The relationship between work in raw bagasse dust and the development of the symptoms seems clear. The close association between the patient's

work and the characteristic clinical picture, with the absence of the picture under other circumstances, points to a definite cause and effect relationship between exposure to the dust and the development of symptoms.

Various causes have been suggested. Jamison and Hopkins[1] isolated a fungus from the first patient reported. In one instance reported by Sodeman and Pullen[5] monilia were found in the sputum but were considered contaminants. Attempts to isolate a fungus from the sputum have not met with uniform success.[2,3] Bagasse itself is difficult to study. After lying in the field for variable lengths of time, with fermentation and fungous and bacterial invasion taking place in the bales, bagasse is a complex substance not easy to analyze. Fungous disease of the lungs of occupational origin is well known, as examplified by the maple-bark disease.[8] Exposure to dust after processing has not yet been shown to produce the picture, and such processing includes heat treatment which would destroy living agents. Then, too, fungous infection, arising from bagasse, has been demonstrated by Morales-Otero and Koppisch.[9] These observers in 1933 reported an epidemic in young chicks exposed to bagasse. The disease was characterized by dyspnea, somnolence, drooping of the wings, loss of weight and fluffing up of the feathers, and death occurred in five days to two weeks. Pulmonary changes were noted in which *Aspergillus fumigatus* was found both in lung sections and by isolation. These authors were able to reproduce the disease. In 1946 Gerstl, Tager and Marinaro[10] also reported the isolation of *Aspergillus fumigatus* as well as other organisms from raw bagasse and were able to reproduce in rabbits and guinea pigs, by both intravenous and intratracheal inoculations, a pulmonary disease in which *A. fumigatus* was recovered. There is the possibility that these pictures represent the counterpart of bagasse disease in humans. Comparisons of the lesions in these animals with those in human lung are now being made by Gerstl and his co-workers. However, in bagasse disease in humans, no fungus has been consistently isolated and there is as yet little evidence to support a fungus as the causative agent. Nevertheless, an infectious theory cannot be disproved on negative or indirect grounds.

The picture is unlike that of byssinosis[5] as it occurs in workers with low-grade stained cotton. An allergic theory has been proposed[2] and has been discussed at length elsewhere.[5] Tuberculosis, suspected especially because of the roentgen appearance of the lungs, has been eliminated as the etiologic agent.

Pneumoconiosis is a possible cause. Bagasse contains five to seven per cent silica (silicon dioxide), but the clinical picture, including the resolution of the process, is unlike that of silicosis.

Sodeman, Pullen, Pinto and Pearson,[6] in experimental studies with bagasse, have shown reactions in the peritoneum of the guinea pig indicating the relative unimportance of the silica fraction. Sodeman and Pullen[5] have demonstrated, in biopsies of the lung during the height of the clinical picture, as well as in a section of lung taken from patient No. 1 of the present report,[4] spicules of a foreign material having the characteristics of bagasse and rotating polarized light as bagasse particles do. This evidence indicates that particles of bagasse do enter the alveolar regions and may initiate a reaction. However, the manner in which such particles may produce the changes remains obscure. Mechanical irritation alone, a reaction of the lung to a chemical product in the bagasse, as well as the introduction of a yet unrecognized infection, are all possibilities.

SUMMARY

Report of four additional cases of bagasse disease of the lung re-emphasizes the characteristics of the clinical picture. Review of the evidence concerning the etiology of this picture indicates that the cause remains obscure.

RESUMEN

Se informa sobre cuatro casos adicionales de la enfermedad pulmonar de bagazo, que recalcan de nuevo las características del cuadro clínico. El repaso de los datos pertinentes a la etiología de este cuadro indica que todavía queda oscura la causa.

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Discussion

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One of the first things that impressed me in reading about this affliction was the importance of history taking, which is mighty important in tracing down a baffling diagnostic problem. If a patient comes to your office with cough and dyspnoea, you might ask him what work he does. If he says he has recently worked in a sugar cane factory you may have the solution of your problem. If you know what things to suspect, you have gone a long way on the road to a diagnosis.

I am not aware of ever having seen a case of bagasse disease, but, neither have I seen George Washington, but still I know a lot about him. I feel confident now, however, thanks to Dr. Sodeman, that, if a bagasse victim came to my office I would make a correct diagnosis.

I was just thinking that it is about time to discover a disease that is curable. When we consider the present sick world with all its devastating afflictions, it's refreshing to be presented with a disease of which you can say with due dignity to the patient, "This, and not you, will pass away."

The continued search for new diseases is as vital to dynamic medicine as is the pursuit of new inventions in industry. By discovery of new diseases I mean, of course, mostly the unveiling of old afflictions in which the etiology, diagnosis and treatment have been on a speculation basis. History is full of instances where darkness clouded the true causative agent of disease. Pulmonary tuberculosis was once believed to be due to irritating secretions falling from the brain to the lungs. Appendicitis was considered as an intestinal colic. These are but brief examples of wise investigators, not satisfied with prevailing beliefs, tearing assunder theoretical speculations and putting truth in their places.

Let us not underestimate the importance of such contributions to medical knowledge that our main speaker has made. It marks another milestone along the road to better medicine and, therefore, improved humanity.

Syphilitic Heart Disease

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Syphilitic heart disease is a misnomer, for the basic lesion in syphilitic heart disease is an aortitis. This, in turn, consists of a perivascular lymphocytic infiltration of the vasa-vasorum in response to the invasion of the spirochete. Depending upon the extent and the degree of this infection within the aorta, we may have, complicating the basic lesion of aortitis, either aortic insufficiency, coronary ostial closure or aneurysm formation. It is most unusual to find a gummatous change within the myocardium or a diffuse lymphocytic infiltration of the myocardium. Rarer still is it to find in association with syphilitic heart disease myocardial infarction as a result of coronary ostial closure.

The diagnosis of aortitis per se is very difficult. It depends upon the history of syphilis or the finding of a positive Wassermann reaction. If the patient is under forty years of age, there should be a tambour aortic second sound and there should be no other complicating cardiovascular disease. The aorta should be widened upon roentgenographic or fluoroscopic examination. In addition, a widening of manubrial dullness and retromanubrial pain may also be present. Any combination of any three of these factors suggests the diagnosis of luetic aortitis. If we hold, however, to these criteria, we seldom make the diagnosis correctly. In fact, we more often diagnose the disease by simply stating that any patient with syphilis has aortitis. Because of the difficulty in the diagnosis of syphilitic aortitis, the material used in this study was selected from those cases of luetic heart disease that were proven at necropsy. In a series of essentially 10,000 necropsies performed by Dr. John F. Noble at the Ancker Hospital, there were 161 patients with proven syphilitic aortitis. Of this group 106 were white males, twenty-five of whom were under the age of fifty years; twenty-eight were white females, twelve under the age of fifty years (Fig. 1); twenty-one were colored males, ten under the age of fifty years; and eight were colored females, three under the age of fifty years. In the necropsy series at Ancker Hospital the ratio of white to colored necropsies is essentially 4.6 to 1 which suggests that syphilitic aortitis in the colored people has about the same incidence as that of the white people.

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Of this group of 161 individuals ninety-six had four plus blood Wassermann tests. Of the nienty-six, forty-nine had indeterminate spinal Wassermann tests in the sense that they were not completely positive; twenty-nine had four plus spinal Wassermann reactions, and the Wassermann test was negative in seventeen individuals. In forty-five of the 161 patients the blood Wassermann was indeterminate in that it was not completely positive. In this group the spinal fluid Wassermann in forty-three individuals was indeterminate and in two the Wassermann test was positive. There were nineteen of the 161 patients with negative blood Wassermann reactions eleven of whom had indeterminate spinal fluid Wassermann tests, two had four plus spinal fluid Wassermann tests and six had negative spinal fluid Wassermann tests. Contrary to the ordinary belief, syphilitic aortitis seems to be associated not infrequently with central nervous system syphilis, and the presence of central nervous system syphilis does not exclude cardiovascular syphilis.

In this group of patients with luetic aortitis, coronary ostial closure was rather common. It involved the right coronary artery in sixty-nine patients with complete closure of the ostia in eight patients, and in fifty of the sixty-nine patients the left coronary artery was also somewhat involved, but to lesser extent. The left coronary ostia was more involved in thirty-nine patients and there was complete closure in two. In this group the right coronary artery was also involved, but to a lesser extent. Valvulitis occurred

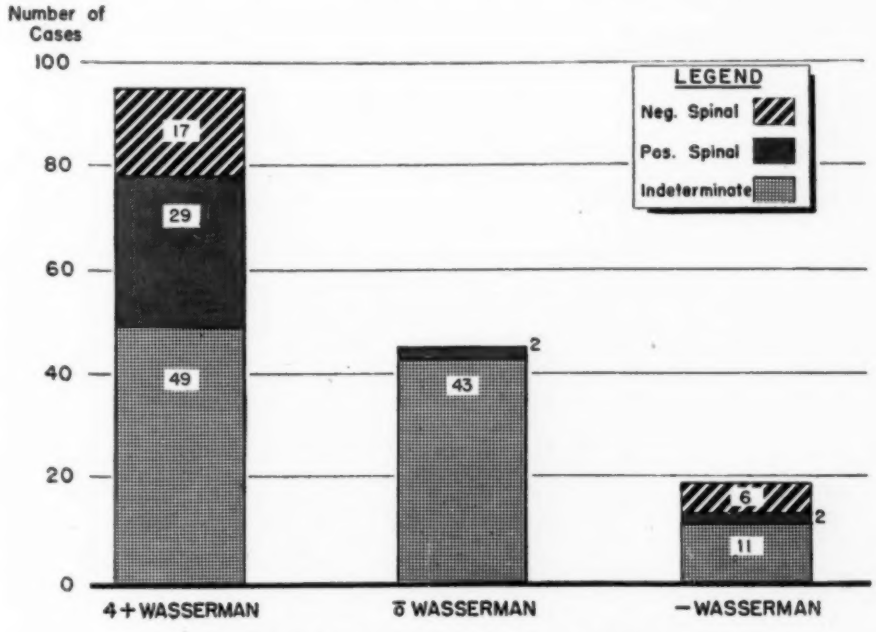

FIGURE 1: Wassermann Test Results.

in eighty-five of the patients. Of eighty-five, thirty-two had valvulitis alone without aortic insufficiency, and in fifty-three individuals aortic insufficiency was present. There were eighteen patients who had miscellaneous complications in association with aortitis such as rheumatic heart disease, auricular fibrillation, hypertension and subacute bacterial endocarditis. In nineteen of the patients aortic aneurysm was present, and in fourteen of these the location of the aneurysm was described. Since the aneurysm involved more than one part of the aorta, the description of their location (Figs. 2 and 3) would make it appear that there were more than fourteen aneurysms. The ascending aorta was involved eleven times, the transverse portion of the aorta five times, and the descending aorta five times. There were two aneurysms of the abdominal aorta, one involved the innominate artery and one the basal artery of the brain. Of the nineteen patients who had aortic aneurysms, fourteen had positive Wassermann tests, two had negative Wassermann tests, and in three the blood Wassermann was not recorded. In ten cases the aneurysm was an accidental discovery and there were no complications. In six cases the aneurysm ruptured, three times producing a cardiac tamponade, twice a bleeding into the pleural space, and in one case the rupture was retroperitoneal. In two of the patients there was a partial rupture of the aneurysm manifested by a superior mediastinal obstructive syndrome. In one patient there was an erosion into the esophagus which was characterized by the spitting of blood.

In thirty-seven cases the clinical findings and histories of rheu-

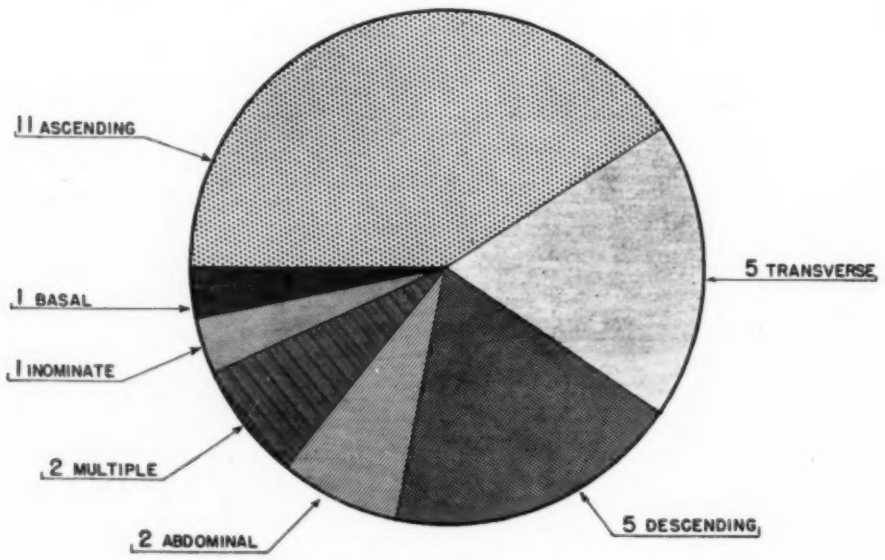

FIGURE 2: Location of aortic aneurysms (14 cases).

matic fever suggested the possibility of rheumatic heart disease. In these patients the correct diagnosis of luetic heart disease was made twenty-eight times and the correct diagnosis of an associated rheumatic heart disease was made twice. In five instances the patient was thought to have rheumatic heart disease when he had luetic heart disease, and in two instances he was thought to have syphilitic heart disease when the necropsy proved that he had luetic heart disease.

Of the entire series of patients it is interesting to note that syphilitic heart disease was the major cause of death in forty-one of the patients, and the causes of death from the anatomical standpoint were combinations of valvulitis, aneurysm and coronary ostial closure. In fifteen per cent of the patients or twenty-four individuals of the group, it was felt that the syphilitic aortitis with its complication played a contributing role in the cause of death. Here it was a combination of valvulitis and aneurysm formation. In ninety-six of the patients the syphilitic artery disease had no connection whatsoever with the cause of death. It was purely an accidental finding.

Rheumatic valve defects were present in fourteen patients. The aortic valve was involved five times, the mitral valve eight times, and the tricuspid valve was involved once. Bacterial endocarditis occurred in five individuals. It was implanted upon a previous

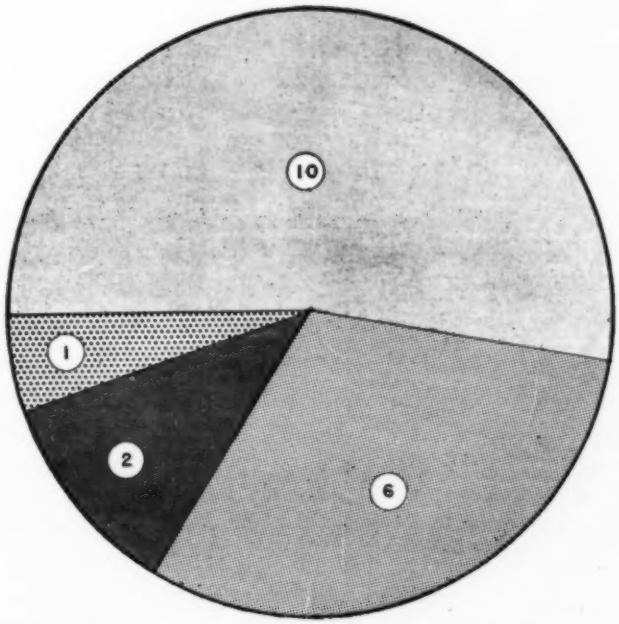

FIGURE 3: Complications in aneurysms (Total of 19 cases).

1 Erosion
2 Partial Rupture
6 Ruptured
10 Uncomplicated

rheumatic valve defect in four of the patients, and in only one instance was it upon a luetic defect. Auricular fibrillation occurred eight times, and in all of these patients some other complicating cardiac disease was present such as rheumatic heart disease or hypertension. Myocardial infarction as a result of closure of the coronary ostia occurred in three of the patients. In two of these individuals death was sudden. In one of the patients death did not occur until a clinical diagnosis had been made.

SUMMARY

Of the 161 patients with syphilitic heart disease the following clinical facts were obtained:

1) In the diagnosis of syphilitic heart disease, one should obtain a positive Wassermann test or the history of syphilitic infection.

2) If we assume that the presence of a positive spinal fluid indicates an infection in the central nervous system, then, contrary to popular belief, central nervous system syphilis is rather common in syphilitic heart disease.

3) When the problem arises as to the etiology of the aortic valve defect, the following facts are important:

(a) Whenever the physical findings are those of a stenosing lesion, then the lesion is rheumatic.

(b) In the presence of a regurgitating type of lesion, the evidence seems to indicate that the lesion is probably luetic.

(c) If we have a diastolic murmur at the apex in the presence of a frank aortic insufficiency and the heart is enlarged to the left, the diagnosis of mitral stenosis even in the presence of a history of rheumatic fever is hazardous. On the other hand, a diastolic murmur at the apex, in the presence of a frank syphilitic aortic insufficiency with the history of rheumatic fever and with the left ventricle not being enlarged, usually means an associated rheumatic mitral valve defect. The diagnosis is further supported if there is an associated auricular fibrillation.

4) Subacute bacterial endocarditis is rare in syphilitic heart disease, yet in five patients this phenomenon did occur. In four, the lesion was superimposed upon an associated rheumatic valve defect and in only one case upon a pure luetic valve defect.

5) The popular opinion that auricular fibrillation is unusual in syphilitic heart disease is widely held and yet, there were eight instances of patients suffering from syphilitic heart disease with auricular fibrillation. In every instance, however, the auricular fibrillation was associated with some complicating factor such as hypertension or rheumatic heart disease.

The results of the analysis of 161 cases of syphilitic heart disease

have been tabulated. While the number is too small to be statistically significant, the analysis presents the pattern of the disease as it was seen at the Ancker Hospital.

RESUMEN

En un estudio de 161 pacientes con enfermedad sifilítica del corazón se obtuvieron los datos clínicos siguientes:

1) Para diagnosticar la enfermedad sifilítica del corazón se debe obtener una prueba de Wassermann positiva o la historia de infección sifilítica.

2) Si damos por sentado que la presencia de un líquido céfalorraquídeo positivo indica una infección del sistema nervioso central, entonces, aunque contrario a la creencia popular, la sífilis del sistema nervioso central es bastante común asociada con la enfermedad sifilítica del corazón.

3) Cuando surge el problema de la etiología del defecto de la válvula aórtica, son importantes los siguientes hechos:

(a) Cuando quiera que los hallazgos físicos indican una lesión estenosante, la lesión es reumática.

(b) Cuando existe una lesión de tipo de insuficiencia, la lesión es probablemente sifilítica.

(c) Si se descubre un murmullo diastólico sobre el ápice cuando existe una insuficiencia aórtica franca y está hipertropiado el corazón hacia la izquierda, es dudoso el diagnóstico de estenosis mitral aún si hay historia de fiebre reumática. Por el contrario, un murmullo diastólico sobre el ápice, cuando existe insuficiencia aórtica sifilítica franca y se obtiene la historia de fiebre reumática y no está hipertrofiado el ventrículo izquierda, generalmente indica un defecto asociado de la válvula mitral de origen reumático. La presencia de fibrilación auricular apoya aún más el diagnóstico.

4) La endocarditis bacteriana subaguda es rara en la enfermedad sifilítica del corazón; sin embargo, ocurrió este fenómeno en cinco pacientes. En cuatro de ellos la lesión estaba superimpuesta sobre un defecto valvular reumático asociado y sólo en un caso estuvo superimpuesta sobre un defecto valvular puramente sifilítico.

5) La opinión popular de que la fibrilación auricular es rara en la enfermedad sifilítica del corazón es muy general; pero, a pesar de eso, hubo ocho casos de pacientes que padecían enfermedad sifilítica del corazón con fibrilación auricular. Sin embargo, en todos los casos la fibrilación auricular estuvo asociada con algún factor complicante tal como hipertensión o enfermedad reumática del corazón.

Pulmonary Mycoses*

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The signs and symptoms caused by pulmonary mycoses are very much like those caused by pulmonary tuberculosis. The x-ray appearance of the lesions may simulate closely that found in tuberculosis. This explains in part frequent diagnostic errors. Hemoptysis and cough with thoracic discomfort are the most prominent symptoms. The diagnosis of mycosis can be established only by careful evaluation of the laboratory methods for the demonstration of fungi in the sputum, gastric contents, and drainages from sinuses and abscesses.

The collection of the sputum should be done as soon after awaking in the morning as possible. Before this is done, however, the teeth should be brushed, the throat gargled, and the mouth and thorat rinsed. Every effort should be made to obtain only sputum from the lungs. Sterilized Petri dishes serve best for collection purposes. The fungi will most often be found in particles or flecks which are usually yellowish or gray in color. Freshly obtained specimens should be sent to the laboratory as soon as possible, where it is examined from direct smears or cultured or injected into animals. Methods and procedures are described in detail by Kurung.[1]

Pulmonary Actinomycosis

The term actinomycosis is applied to two groups of diseases of man and animals. In one group anaerobic Actinomyces bovis with the characteristic "sulphur granules" are observed in pus or tissues. They may be found in the gums and tonsils[2,3] of apparently healthy normal individuals. The second group includes aerobic Actinomyces, now called Nocardia,[4] a filamentous branching organism without any radial arrangement and without granule formation. Two recognized species of this group are: Actinomyces graminis which is non-acid-fast and Actinomyces asteroides which is acid-fast. Proved cases of infection with Actinomyces graminis are rare. Pathogenic Nocardia have been isolated from the soil.[5] In general the sputum is thick, tenacious and grayish yellow in color with almost no odor. Frequently it contains no fungi, and they have to be obtained from "cold abscesses" and sinuses or microscopic examinations of tissue. The drainage from fistula has

*Read before the Fulton County Medical Society, December 4, 1947.

no odor. Local or generalized adenitis may be present. Laboratory examination of the blood usually shows a low hemoglobin and red cell count. The differential count shows an average of 70 to 80 per cent leucocytes, 2 to 10 per cent eosinophiles and 10 to 18 per cent lymphocytes.

Roentgenograms may show any grade of infiltration of the parenchyma and involvement of the hilar glands. Radiographic changes take place slowly and are usually followed by pleural involvement.

Treatment with penicillin has proven helpful in most cases, but many are recognized too late for this antibiotic to be effective. Iodine and x-ray therapy are of doubtful value. Strict bed rest, general supportive treatment, and cleanliness are also necessary aids.

An interesting entity known as farmer's lung has been reported.[6] It is clinically and radiologically a fairly well characterized disease. The usual history is that a farmer, after working continuously for a time with mouldy hay from which a steamlike cloud of fungus-laden dust arises, develops a short dry cough with little or no expectoration; he may also suffer from general malaise, weakness and slight fever, but the outstanding feature is distressing dyspnea provoked by physical exertion. Removal from exposure to mouldy hay dust is followed by rapid recovery, which is usually complete and permanent if further exposure is avoided. With successive seasonal recurrences the disease tends to progress to chronicity; the cough becomes more productive, the abundant sputum is tenacious and frothy or purulent and slight hemoptysis may occur; physical weakness is marked and the dyspnea severe. At this stage, the lung may show fibrosis, emphysema, and possibly bronchiectasis, and the x-ray picture is usually characteristic. The relation of farmer's lung to inhalation of mouldy hay dust is unquestioned. The commonly held view that it is a true pulmonary mycosis is based on the occupational history of the patient and the cultivation of moulds from his sputum. The anaerobic species Actinomyces bovis was cultivated from the sputum in 2 cases. In one of these the lesion appeared to be confined to the lung, but in the other case the lung infection, complicated by empyema, was part of a generalized disease which proved to be fatal.

Pulmonary Coccidioidomycosis

A full historical report on this disease was made by Bass, et al.[7] It is caused by Coccidioides immitis. A detailed epidemiological investigation was carried out in four army air fields in the San Joaquin Valley, California by Smith and Beard.[8] The investigation was based on coccidioidin testing permanent personnel and

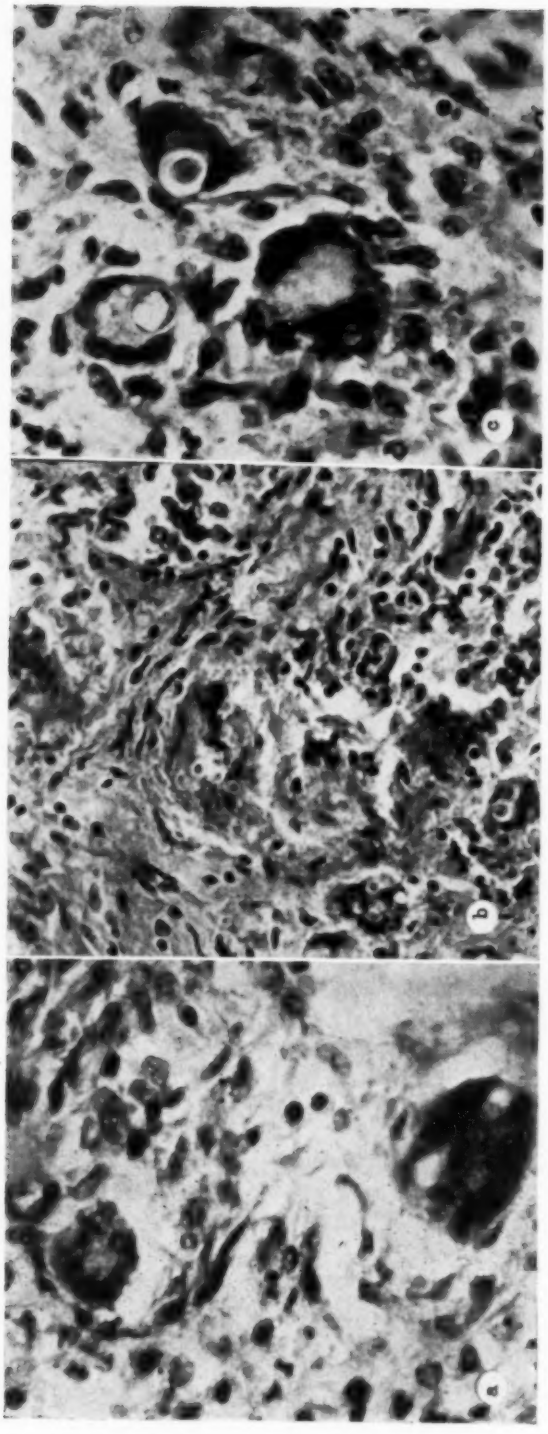

FIGURE 1

(a) Lung—Blastomycosis, Giant Cell (b) Lung—Blastomycosis x950 (c) Lung—Blastomycosis x100

at first annually, later semi-annually, retesting the negative reactors. The pattern of geographic distribution of coccidioidal endemicity showed an increase in the southern part of San Joaquin Valley with a decrease as one proceeds northward. Maximal incidence occurred in the arid dusty summer and autumn months. The seasonal infection rate showed that dust was the mechanism by which the fungus was transported. Grassing, paving roads and runways, and ultimately the use of highly refined oil on atheletic areas were important local dust control measures. This control reduced infection rates from one-half to two-thirds.

Texas, Arizona, New Mexico, California and Argentina contain endemic areas. Arizona Indian children, living on reservations, have shown as high as 90 per cent positive reactions to the cocidioidin skin test. The author of this paper was also stationed in the San Joaquin Valley and saw large numbers of these cases in the terminal stage. The fatal cases are as tragic as death from malignancy.

The initial infection (incubation period, 5 to 20 days) is usually asymptomatic, but when symptomatic, the onset is sudden and comparable with influenza as; chills, fever, pleural pain, hemoptysis, sore throat, malaise, joint pain, headache, and cough (productive or nonproductive). The erythema nodosum and multiforme types of rash may be seen from the fifth to fifteenth day of the disease.

The secondary or disseminated form is frequently accompanied by subfebrile temperatures and great loss of weight. This form is found more often in the Oriental and dark-skin races, and in these races the mortality rate is much higher. Dissemination through the blood and lymph channels causes any of the organs of the body to be involved.

Positive diagnosis in both forms is made by finding the fungus by smear or animal innoculation. Other aids are: a history of exposure in one of the endemic areas, and a positive skin test to a coccidioidin intradermal injection. This skin test should be considered in the same way and of the same value as the tuberculin skin test. Positive serological tests for precipitins and complement fixation are also helpful. A constant rise in titer suggests dissemination of the infection. Sensitivity is determined by the intracutaneous injection 0.1 cc. of a 1:100 dilution of standard coccidioidin. A negative coccidioidin test to a 1:10 dilution is not unusual in the terminal stage of the infection.

Jamison[9] reports the predominant roetgen manifestations in coccidioidal pulmonary infections which persisted for months or years following the acute initial phase of the disease as follows: (1) nodular parenchymal foci; (2) cyst-like cavities; (3) persistent

pneumonitis; (4) mediastinal and hilar adenopathy; (5) pleural effusion; (6) miliary lung involvement, metastatic bone foci and other evidences of dissemination. Negroes are about 100 times more likely to develop fatal disseminated coccidioidomycosis than white people. White persons are more apt to develop a meningeal type of coccidioidal dissemination, while Negroes show a higher incidence of subcutaneous abscesses. Dissemination, when it occurs late, appears to be of less ominous prognostic significance than when occurring within the first few days or weeks of the initial acute illness.[10]

No specific treatment is of value. Persistent cavity formation and hemorrhage are handled in the same manner as in tuberculosis. Iodides, thymol, penicillin, copper and immuno-transfusions have all failed. General supportive treatment should be used in every case. Desensitization is difficult but should be tried and followed by iodide or colloidal copper.

Pulmonary Blastomycosis

Pulmonary Blastomycosis ("Gilchrist's disease") is usually a manifestation of a generalized fungus infection caused by Blastomyces dermatitidis which is more commonly found in the skin. They occur in the lesions, pus, and sputum and appear as gram staining oval or round, thick walled bodies (7 to 10 micra) with fine granular protoplasm (Fig. 1).

Usually the onset is insidious beginning with a mild respiratory infection. A dry hacking cough, chest pains, slight fever and some dyspnea are usually present. As the disease progresses all symptoms become more severe. Loss of weight and strength become apparent. Signs of toxemia or night sweats appear. Later all organs of the body may be involved. X-ray films usually show dense masses with irregular outlines. Often the involvement of a rib at the same location suggests carcinoma. Rarely early parenchymal lesions may be minimal with marked enlargement of the mediastinal nodes.

The disease is often chronic in nature lasting for years in its cutaneous form, but in the pulmonary of systemic form, death usually occurs in 6 months to 3 years.[11,12] A fatal outcome is expected in those having a high antibody titer (indicating extensive disease) and a negative or slightly positive skin test. The prognosis is best in hypersensitive patients without complement fixing antibodies.

Patients with definitely positive skin tests should be desensitized with vaccine or antigens before iodides are administered since a failure to desensitize may result in a lack of improvement or even a prompt exacerbation of the infection.[13] Patients with

slightly positive skin tests may be successfully treated with large doses of iodides. Stock or autogenous vaccines should be administered until the patient has complement-fixing antibodies in the serum before administering iodides.

Pulmonary Sporotrichosis

Sporotrichosis is a chronic fungus infection of the skin but does occur in the lungs. It is caused by Sporotrichum Schenckii, which is primarily a saprophyte or parasite of plants.[14] It is most commonly found in farmers, laborers and horticulturists. The pulmonary form is a manifestation of the disseminated type. Prognosis in this type is poor. Iodides are beneficial but must be continued long after all clinical symptoms have disappeared. X-ray therapy, surgery and vaccines are also used.

Pulmonary Conisporiosis

Smith[13] states that a peculiar type of pneumonitis, presumably allergic, occurs in laborers in the Northern forests who are repeatedly exposed to inhalation of spores of Coniosporium Corticale growing on the inner bark of maple logs. Recovery results when patient is removed from contact with the spores.

Pulmonary Moniliasis

Probably the most common fungi are those which belong to the Monilia group. Treatment is difficult to evaluate because: (1) the fungi appear to "go and come." Long periods elapse when they cannot be found, only to reappear both before and after treatment is started. (2) Monilia is usually considered to be an insignificant saprophyte in the respiratory tract. The author has seen numerous cases that have been pathogenic and caused acute illness. They may be the cause of bronchitis,[15] pneumonitis,[16-18] meningitis,[19] endocarditis,[20,21] and osteomyelitis.[22] (3) Many cases with fungus disease are hypersensitive to iodine which is the drug of choice. (4) The failure to determine the degree of sensitivity of the patient, and therefore, the failure to desensitize prior to iodides is another cause of ineffective treatment.

Bronchial infection sometimes disappears spontaneously, but often lasts for years with periodic progressions and retrogressions. X-ray films show nothing or slight peribronchial thickening and occasionally linear fibrosis. Parenchymal involvement is more rare and more serious than a bronchial infection. Elevated pulse and temperature, pleural pains, and an occasional effusion is present. The cough is severe and productive of mucoid, gelatinous sputum. A purulent sputum indicates a secondary infection. Pneumonic consolidation with the usual signs and symptoms may develop.

Death may occur when two or more lobes are involved with a pneumonic process.

Hiatt and Martin[23] report a patient from whom Candida albicans was isolated from the sputum on numtrous occasions. The patient was sensitive to iodides, and gentian Violet therapy caused an unfavorable reaction. The patient was treated with increasing doses of the immune rabbit serum and made a dramatic recovery.

Thrasher's lung[24] is caused by monilia and is characterized by a sudden onset with chill, fever, malaise, cough, and mucopurulent or bloody expectoration. Dyspnea is the most important and persistent symptom. Fine bronchial rales are heard over the bases, a slight leucocytosis is usually present, and the sedimentation rate is increased. Roentgenograms reveal pictures similar to miliary tuberculosis, but the fine mottling is densest in the parahilar regions and the bases, while the apices are relatively free. The x-ray signs may disappear or may result in patchy fibrosis with bronchiectasis. Similarities with sarcoidosis are especially to be noted, particularly in the presence of negative tuberculin reactions. Potassium iodide seemed to be of benefit, particularly in the milder cases, especially if the patients have not developed a hypersensitivity. Hypersensitive patients should be desensitized and treated cautiously. Intravenous gention violet[25] has been used successfully in some acute pulmonary cases.

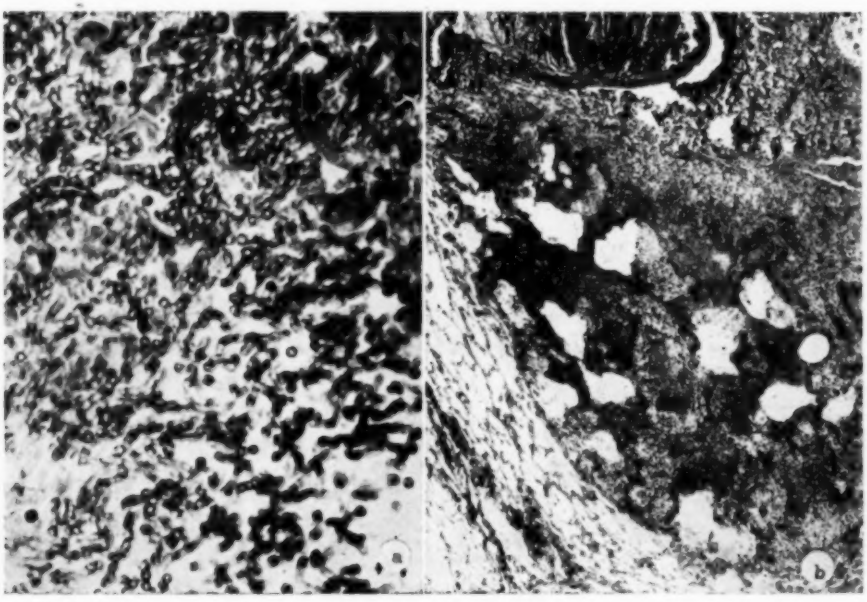

FIGURE 2

(a) Lung—Aspergillosis x700.

(b) Lung—Abscess. Aspergillosis x30.

Pulmonary Aspergillosis

Squab feeders, fur cleaners and agricultural workers are especially prone to have the infection. Primary pulmonary aspergillosis is difficult to diagnose. Symptoms, signs, and clinical courses resemble pulmonary tuberculosis. However, aspergilli frequently are isolated from the sputum of patients with chronic bronchitis, intrinsic asthma and appear to be an accidental contaminant which gives no symptoms (Fig. 2).

X-ray films may show smooth, dense lesions and cavity formation or they may show nodular diffuse shadows.

Iodides have been used with success. Hypersensitive patients should be desensitized. For the sake of completeness, penicilliosis and mucormycosis should be mentioned as similar to aspergillosis. Also, reference should be made to a case of pulmonary trichophytinosis reported by Denis, Hendtlass and Martin.[26]

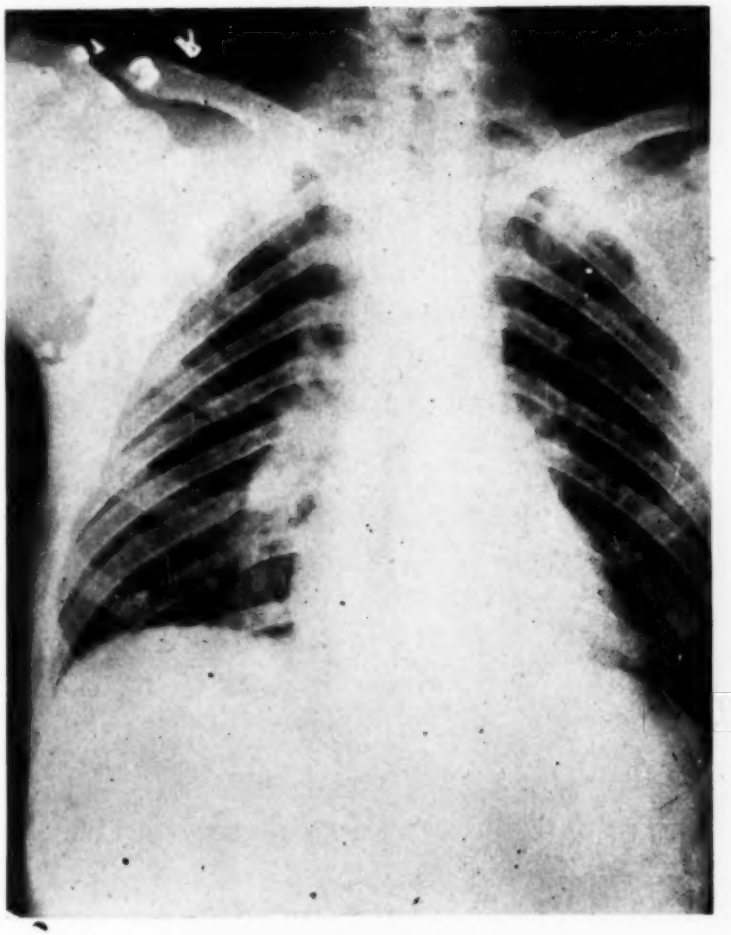

FIGURE 3: Torulosis—X-ray of Lungs.

Another rare fungus is Geotrichum which is described by Kuntstadter, et al,[27] who state that the genus is mostly saprophytic, but infections have been reported in man. This fungus causes symptoms of chronic pulmonary infection. Roentgenograms show peribronchial thickening and fine mottling in the mid-lung fields or in the bases. More severe cases present dense patches of infiltration with or without cavity formation. Iodides and vaccines are usually effective.

Pulmonary Torulosis

Pulmonary Torulosis or Cryptococcosis is one of the fungus infections which has a tendency to invade the central nervous system. These patients may develop a generalized infection. Mildness may mask the infection for many months, and the only symptom may be a slight cough.

X-ray films in primary pulmonary infections show dense shadows resembling far advanced pulmonary tuberculosis or neoplasm (Fig. 3). Cavities are not usually found nor is the mediastinum usually involved. Later miliary lesions may be present. The author has used iodides and noted that the acute initial stage subsides into a quiescent stage which may flare up six months or a year later in an entirely different part of the body, finally terminating in central nervous system invasion. Reeves, Butt and Hammock,[28] and Smith[13] report a cure with sulfadiazine.

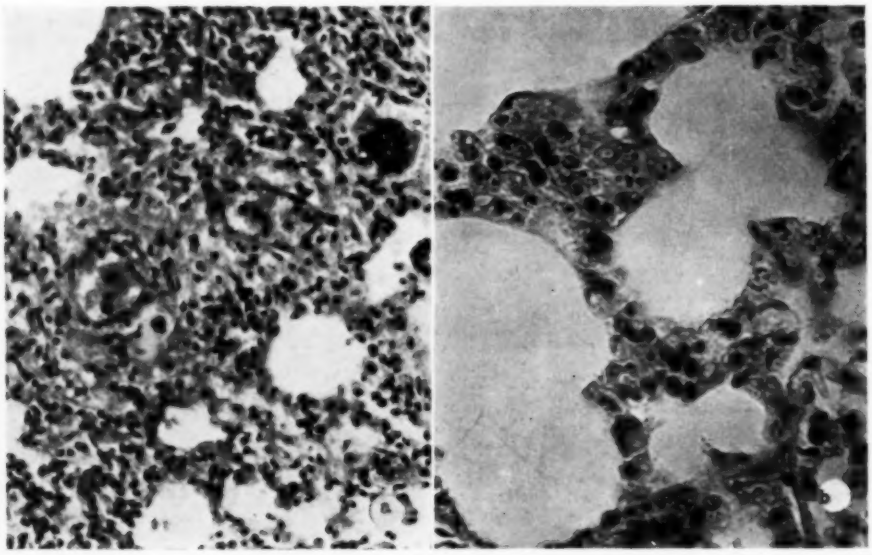

FIGURE 4

(a) Lung—Histoplasmosis x330.
(b) Lung—Histoplasmosis x810.

Pulmonary Histoplasmosis

The status of Histoplasma capsulatum, as related to pulmonary calcification in the human is still questionable. It is found in the cytoplasm of endothelial and mononuclear cells.[28] The Tuberculosis Control Division of the U. S. Public Health, in its annual report of 1946, mentions that such infections are probably responsible for pulmonary calcification observed in tuberculin negative reactors.

Young children are very susceptible to the disease which is characterized by fever, emaciation, anemia, leukopenia, splenomegaly and hepatomegaly. The literature[30] shows that one-third of the reported cases began in the ear, nose, lips, mouth, pharynx and larynx. Pulmonary manifestation was thought to be found usually in the disseminated or generalized cases but Smith,[31] Christie, Peterson,[32] and Palmer[33,34] have accumulated evidence that there may be a benign or primary pulmonary form that has been unrecognized which ultimately may result in pulmonary calcifications (Fig. 4). In the disseminated stage, histoplasmosis is fatal. Treatment is unsatisfactory.

CONCLUSIONS

The author has noted the frequent association of allergies with mycoses. Iodides are often not tolerated well by these patients. Chronic types of dermatitis are frequently aggravated.

Failure to desensitize hypersensitive patients, and the failure to establish complement-fixing antibodies in the patient are responsible for many of the failures in treatment of these diseases.

It is important to stress the simularity in signs, symptoms and roentgen findings between pulmonary mycoses and pulmonary tuberculosis. The care in making the correct diagnosis cannot be emphasized too greatly. Laboratory procedures must be easily available and adequate. Much time is required for continued culture of the fungi for correct identification.

CONCLUSIONES

El autor ha notado la frecuente asociación de alergias con micosis. A menudo estos pacientes no soportan bien los yoduros. Frecuentemente se agravan los tipos crónicos de dermatitis.

La omisión de desensibilizar a los pacientes hipersensibles y la falta de descubrir anticuerpos de fijación de complemento en el paciente, son responsables por muchos de los fracasos en el tratamiento de estas enfermedades.

Es importante recalcar la semejanza entre los signos, síntomas y hallazgos roentgenológicos de las micosis pulmonares y la tuberculosis pulmonar. Debe hacerse gran hincapié sobre la necesidad

de hacer un diagnóstico correcto. Se debe contar con adecuados y disponibles procedimientos de laboratorio. Se requiere mucho tiempo para el cultivo contínuo de los hongos a fin de hacer la identificación correcta.

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Mediastinal Defect with Interpleural Communication Observed During Pneumothorax Therapy: Report of a Case*

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The phenomenon of interpleural communication is exceptionally rare,[7,8,9] only ten cases having been reported in the entire medical literature. It should be mentioned, however, that of these, two cases with mediastinal defects were found in patients without any tuberculous infection. One case reported by LeWald[3] in 1926 was of nontuberculous origin, no definite mediastinal defect having been proved; and as that author stated, "Evidently air had passed from the right pleural cavity into the left pleural cavity in some way, apparently through the upper portion of the mediastinum." The second unusual case, described in 1939 by Ochsner and DeBakey,[6] presented a congenital mediastinal anomaly in a child of 18 months of age. They made their diagnosis of a left sided diaphragmatic hernia on the basis of fluoroscopic and x-ray studies; hernioplasty was contemplated. During the operation, however, they found, in addition to a defect in the left diaphragm, a complete absence of the anterior mediastinum. The pleural cavities presented one sac and both lungs could be inspected from a left sided surgical approach by removal of the seventh rib. We must say, therefore, that only eight cases of interpleural defects resulting apparently from tuberculous infection actually remain.

We have recently observed an unusual case with a defect in the anterior superior mediastinum associated with pulmonary tuberculosis in a young white male patient.

Before presenting our case, however, we feel that it is important to call attention to the fundamental anatomy of the mediastinum without going into detail. The weakness of the anterior superior chamber of the mediastinum is explained on the histological basis of the structure there being composed mostly of adipose and loose connective tissues which have replaced the atrophied thymus gland in the adults. Nitsch[5] as far back as 1910 in his studies of the mediastinal structure pointed out two anatomically weak places. One "weak spot" he found posterior to the upper portion of the sternum and the second "weak point" he found in the posterior inferior portion of the mediastinum near the level of the 8th rib.

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The case presented in this paper is almost similar to the cases reported by McCallum,[4] Julien[2] and Dumarest.[1] In our case there was also found a definite interpleural communication during pneumothorax therapy, the patient having very interesting sensations during the passage of the air through his mediastinal defect.

CASE REPORT

A.N.R., No. 49,275, a white male, aged 31, was admitted to the Veterans Hospital, Alexandria, Louisiana, on June 19, 1947, by transfer from another hospital for further treatment of pulmonary tuberculosis.

At the previous hospital the patient was treated for sinusitis and tonsillitis, and during the routine x-ray examination of the chest a minimal tuberculous lesion in the right upper lobe was found. Subsequent roentgenogram (Fig. 1) taken a month later, on February 18, 1947, showed an extension of the tuberculous involvement in the right upper lobe and the sputum examinations were positive for acid fast organisms. Because of the progressive nature of the tuberculous lesion on the right side, positive sputa, and age of the patient, artificial pneumothorax was induced on March 18, 1947, and this was maintained until time of admission to this hospital. During the third refill of air administered to the right side, patient stated that he experienced a sudden pain on the left side equivalent to a sensation of receiving pneumothorax bilaterally. An immediate x-ray examination of the chest disclosed bilateral marginal collapse.

On admission to this hospital, patient was well developed and nourished and did not appear ill. Temperature, pulse and respirations were within normal limits and the blood pressure was 120/82. Physical examination of the chest revealed signs of bilateral pneumothorax; no rales or wheezes were audible. Roentgenological examination of the chest confirmed the physical findings. There was 15 per cent collapse of both lungs with thin apical adhesions on the right side, scattered nodular densities throughout the upper third of the right lung field; no evidence of cavitation. Routine sputum examinations, as well as concentrates and cultures, have been consistently reported negative for tubercle bacilli. Urine and Wassermann examinations were negative. Sedimentation rate was 10 mm. in one hour. Complete blood count revealed: RBC, 4,820,000; WBC, 17,500; Hg., 13.9 grams; Polymorphonuclears, 87 per cent; Lymphocytes, 13 per cent. Repeated white blood count remained high, up to 14,100. Blood chemistry showed normal findings.

It was felt that we were dealing here with one of two conditions: either a congenital anomaly of the mediastinum, or an unusual complication of a tuberculous infection. In either case the pneumothorax given on the right side would have passed through a weak spot or an opening in the mediastinum into the contralateral side. To substantiate the above observation careful fluoroscopic and roentgenographic studies of the chest were made on this patient prior to the pneumothorax refills and subsequent to it. The first x-ray film of the chest, taken July 18, 1947 (Fig. 2), showed about 10 per cent collapse on both sides, and the one following the refill of 300 cc. of air (Fig. 3) disclosed an increase

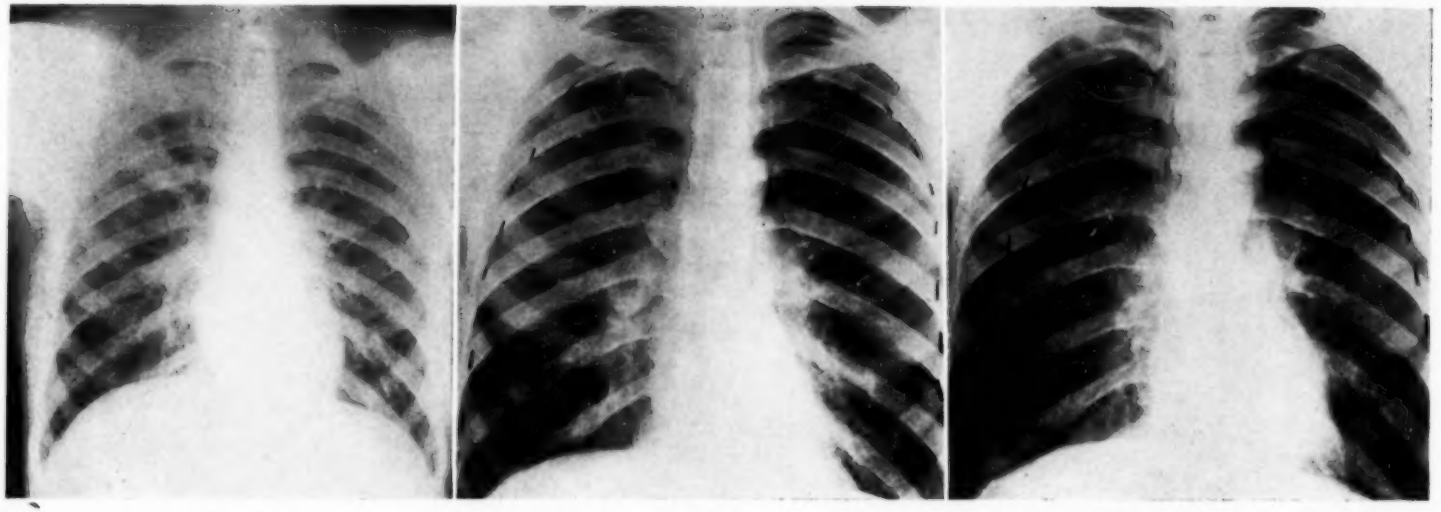

FIGURE 1 FIGURE 2 FIGURE 3

Fig. 1: Chest film, taken February 18, 1947, reveals tuberculous involvement on the right side. The infiltration seems to be exudative in character.—*Fig. 2:* Chest film, taken July 18, 1947, prior to pneumothorax refill, shows only 10 per cent collapse of both lungs. *Fig. 3:* Chest film, taken July 18, 1947, *following* the pneumothorax refill on the right side, shows an increase in the amount of the collapse bilaterally up to 30 per cent.

to 30 per cent bilaterally. To ascertain a to and fro passage of air through the mediastinum a refill was at this time given on the left rather than on the right side, yet here again an increase in the amount of collapse was noted bilaterally.

On careful questioning of the patient for any special sensations in his chest during the time when the air passes from the right pleural cavity into the left side, he volunteered the following information: "About 10 minutes after each refill I feel heaviness and pressure in the upper part of my chest which lasts for about 30 to 50 minutes." During this period, however, the patient did not show signs of difficulty in swallowing or breathing. The fluoroscopic studies of the chest at that time showed a bilateral mediastinal bulge, which disappeared after the pneumothorax equalized in both pleural cavities.

The atmospheric changes have been studied in both pleural cavities, prior, during and following a pneumothorax refill, by inserting simultaneously a needle connected with the pneumothorax machines to each side of the chest. The following manometric changes have been registered: The pleural pressures prior to refill on the right side was −5, −1, and −3, −2 on the left side. After 300 cc. of air was injected into the right side, the manometric readings changed to −7, +2 on the right side and −4, 0 pressure on the left side.

Keeping the needles in position for 10 minutes—during which time the patient was having the pressure sensations in his upper chest—the intrapleural pressures on the right side remained about the same, but on the left side the intrapleural pressure fluctuated more to the negative side to a final reading of −8, 0. It was also observed that patient required frequent refills to maintain a satisfactory collapse.

It is interesting to note the vital capacity changes in this case following pneumothorax refill. The capacity prior to pneumothorax was 2900 cc. while a refill of 200 cc. of air decreased the capacity to 2550 cc.; in other words an unusual decrease of the vital capacity of almost twice the amount of air given.

Comment

The symptoms, physical and roentgenological examinations in this case point unmistakably to a defect in the anterior superior mediastinum. The defect appears to be of other origin than a rupture in the mediastinal walls from increased intrapleural pressures, since the manometric readings were neutral or mostly negative. This is a very interesting case of an interpleural communication resulting in a bilateral pneumothorax. In about 40,000 pneumothorax refills given by the author during the last 10 years

he has never encountered a similar case.

The question naturally arose as to further management of the case. After due consideration it was decided to abandon pneumothorax and keep the patient under close observation as far as his tuberculous lesion was concerned. The reasons favoring discontinuance of pneumothorax were as follows: (a) the lesion in the right upper lobe was of recent nature and exudative in character, (b) all recent sputum examinations were constantly negative for tubercle bacilli, (c) there is the danger of mediastinal emphysema in case of blockage in the passage of the air through the mediastinal opening, and (d) there is the possibility of pleural, and subsequent mediastinal effusion, or empyema, following infection during the course of pneumothorax therapy. Furthermore, in case of progression of the tuberculous infection in the right side a thoracoplasty will be performed just as Smith and Willis[8] did in 1929 in their case.

It is worthwhile to report that the patient at present is in excellent clinical and physical condition and that both lungs are reexpanding without complication.

SUMMARY

1) An unusual case of mediastinal defect without apparent other organic anomalies is presented.

2) Only ten previous cases with similar defects are described in the medical literature.

3) Artificial pneumothorax induced on the right side caused transient bilateral herniation of the mediastinum with collapse of both lungs.

4) There was a definite to and fro passage of air during pneumothorax therapy given on either side.

5) The reasons for discontinuation of pneumothorax are here outlined.

RESUMEN

1) Se presenta un raro caso de defecto mediastínico sin ninguna otra anomalía orgánica aparente.

2) En la literatura médica se han descrito solamente diez previos casos con defectos semejantes.

3) El neumotórax artificial que se aplicó en el lado derecho causó una hernia bilateral transitoria del mediastino con colapso de ambos pulmones.

4) Cuando se dio una insuflación de cualquier lado, el aire pasó de un lado al otro.

5) Se bosquejan aquí las razones por las cuales se descontinuó el neumotórax.

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Cystic Disease of the Lung*

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In the past ten years at least 400 articles concerning cystic disease of the lung have appeared in the literature. Considered rare[1] twenty years ago, it is now a relatively common disease.[2,3]

Much of the past literature deals at length with the origin of pulmonary cysts. In the early literature the condition was considered to be congenital in origin, or at least the result of developmental arrest.[1,2,4] As the years passed, however, it became evident that in many cases the disease was acquired.[5] At present the literature gives the impression that cystic conditions of the lungs are probably more often acquired than congenital.[6,7,8] It is emphasized that the origin makes little difference, except for academic interest.[9] At one time it was thought that epithelization, particularly the stratified columnar type, indicated a congenital origin; now it is believed that in most cases such epithelium may occur in acquired cysts.[9] Because of destruction by infection, epithelium may be absent even in the congenital cysts,[10] therefore it is no longer a reliable criterion of origin. It is generally agreed that given a specific case of cystic disease of the lung, it is impossible to definitely determine whether its origin was congenital or acquired.[11]

Heretofore many names have been given to the condition[4] and at least twenty different terms appear in the literature.[12] Cystic disease of the lung has become the accepted term.[11,13] Thus, the congenital nature or the acquired nature of the origin is circumvented. The numerous individual descriptive names and the many classifications advanced[2,4,11] indicate the desire to segregate and describe different entities that may be cystic in nature. Since the general term, cystic disease of the lung, admittedly a hodgepodge of conditions, is gaining in favor, it would indicate a failure in the endeavor to separate the condition into different entities; it would also indicate the existence of some strong common denominator.

The literature indicates a fairly close agreement upon symptoms and classifies these in the following categories: (a) asymptomatic, (b) symptoms caused by increased pressure within the cyst, (c) symptoms caused by infection of the cyst, and (d) hemoptysis.

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Most patients with pulmonary cysts are in the asymptomatic group.[2,4,14-17]

There is close agreement on the means of diagnosis by roentgenography, bronchography, bronchoscopy, and biopsy if open drainage is performed. In long-standing infection the roentgenogram reveals absence of retraction of the chest wall, lung or mediastinum, and absence of pleural thickening.[3,12] In uncomplicated cases the fluid-filled cyst may be confused with a neoplasm. The air and fluid containing cyst must be differentiated especially from pyogenic abscess and tuberculous cavities. The space containing only air offers less of a problem. However, if the cyst is large enough to fill the hemithorax, the differential point is the finding of lung parenchyma at the cardiophrenic angle and over the medial portion of the diaphragm. If biopsy of the cyst is possible it will reveal epithelium and, perhaps, bronchial structures.

There has been a wide variation in the accepted manner of treatment.[4,15,16] However, recent literature would indicate that extirpation is the procedure of choice and this may be accomplished by enucleation of the cyst, partial lobectomy, lobectomy, or pneumonectomy, depending upon the requirements of the individual case.[4,11,18,19] Infection may necessitate drainage preliminary to excision.

Since 1940 the author has treated fiftten patients with cystic disease of the lung. The range in ages was two months to fifty-six years. There were eight males and seven females. In seven instances the condition was present on the right side and in eight on the left side. The symptoms seemed to follow definite clinical patterns. In three patients the diagnosis was pyogenic lung abscess (Fig. 1); two were treated for eighteen (Fig. 1a and 1b) and twenty-four years (Fig. 1c, 1d and 1e) respectively for chronic lung abscess. There were two cases of acute pyogenic pleural empyema and these were treated by open thoracotomy (Fig. 2). Not until they were thought to be patients with chronic empyema was the true nature of the disease recognized. Two patients developed pleural empyema secondarily to the pulmonary cysts (Fig. 3). Two developed cysts resulting from emphysema (Fig. 4). One was an infant (Fig. 4c, 4d and 4e), age two months, who followed a course as described by Caffey;[20,21] the other had an extremely large cyst that filled the left hemithorax (Fig. 4b) displaced the heart to the right, and then herniated into the right pleural cavity. In two patients the cysts were associated with bronchiectasis (Fig. 1e and Fig. 5b and 5c). One case of cystic bronchiectasis was noted (Fig. 5a). In one instance a cyst completely filled with fluid was diagnosed as carcinoma of the

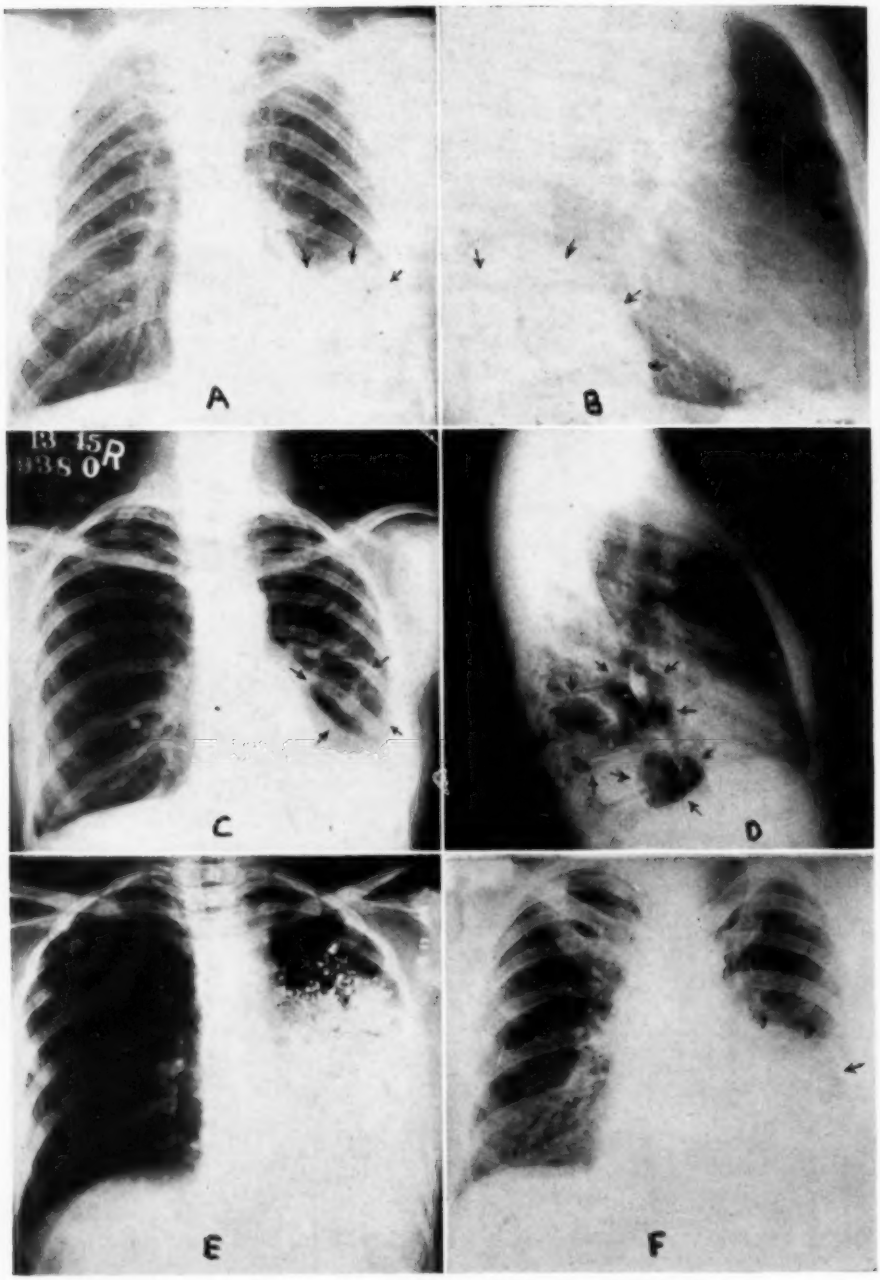

FIGURE 1: Cystic Disease of the Lung Diagnosed as Chronic Lung Abscess.

Fig. 1 A and B, a white male, H. D., age thirty-seven, had an open drainage of a lung abscess or empyema eighteen years before. He continued with symptoms of a mild form consisting of copious purulent expectoration, cough, and occasional episodes of fever. Findings characteristic of a lung cyst and trabeculations with an epithelial lining recently were found at open drainage. The presence of many bronchi opening into the cavity and a history of previous open drainage seem to indicate that it was an abscess cyst.

Fig. 1 C, D and E, a thirty-three year old white woman, M. G., developed

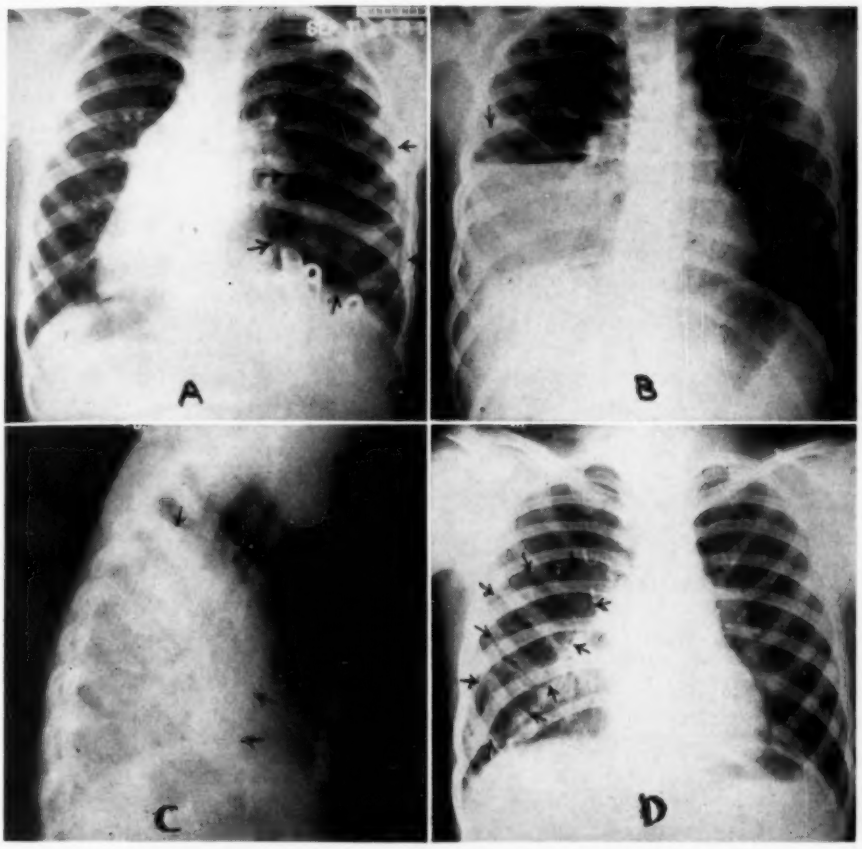

FIGURE 2: Cystic Disease of the Lung First Diagnosed as Acute Empyema and Later as Chronic Empyema.

D. H. was a seven year old white girl. A diagnosis of generalized empyema was made and an open thoracotomy was performed. One year later the patient was referred for treatment of chronic empyema. Roentgenograms (Fig. 2 A) revealed the absence of retraction of the chest wall and thoracic content and absence of pleural thickening. A biopsy led to a diagnosis of lung cyst. Lobectomy of the lower lobe of the right lung was followed by complete recovery.

Fig. 2 B, C and D, was a nine year old white boy, F. D. Except for minor details his disease followed rather closely the same clinical course as D. H. A lobectomy done elsewhere was successful.

A biopsy taken at the initial open drainage of the empyema would have made possible a correct diagnosis for both patients.

FIGURE 1 — Continued:

pulmonary symptoms at nine years of age following a tonsillectomy. An open thoracotomy was performed a few months after the onset. The wound healed but thereafter twice each day she would empty the lung of a large amount of purulent material. During her first pregnancy at the age of thirty-three, severe pulmonary hemorrhages occurred. Because of associated bronchiectasis a pneumonectomy was done and the patient made an uneventful recovery. An epithelial lined cavity with trabeculations and many bronchi leading into it, together with a history of a previous open drainage, seem to indicate that it was an abscess cyst.

Fig. 1 F, a forty-two year old white male, J. N., had a short period of illness. A diagnosis of pyogenic lung abscess was made. At open drainage the characteristics of an infected cyst were noted. Failure to heal was predicted; however, healing was rapid and complete.

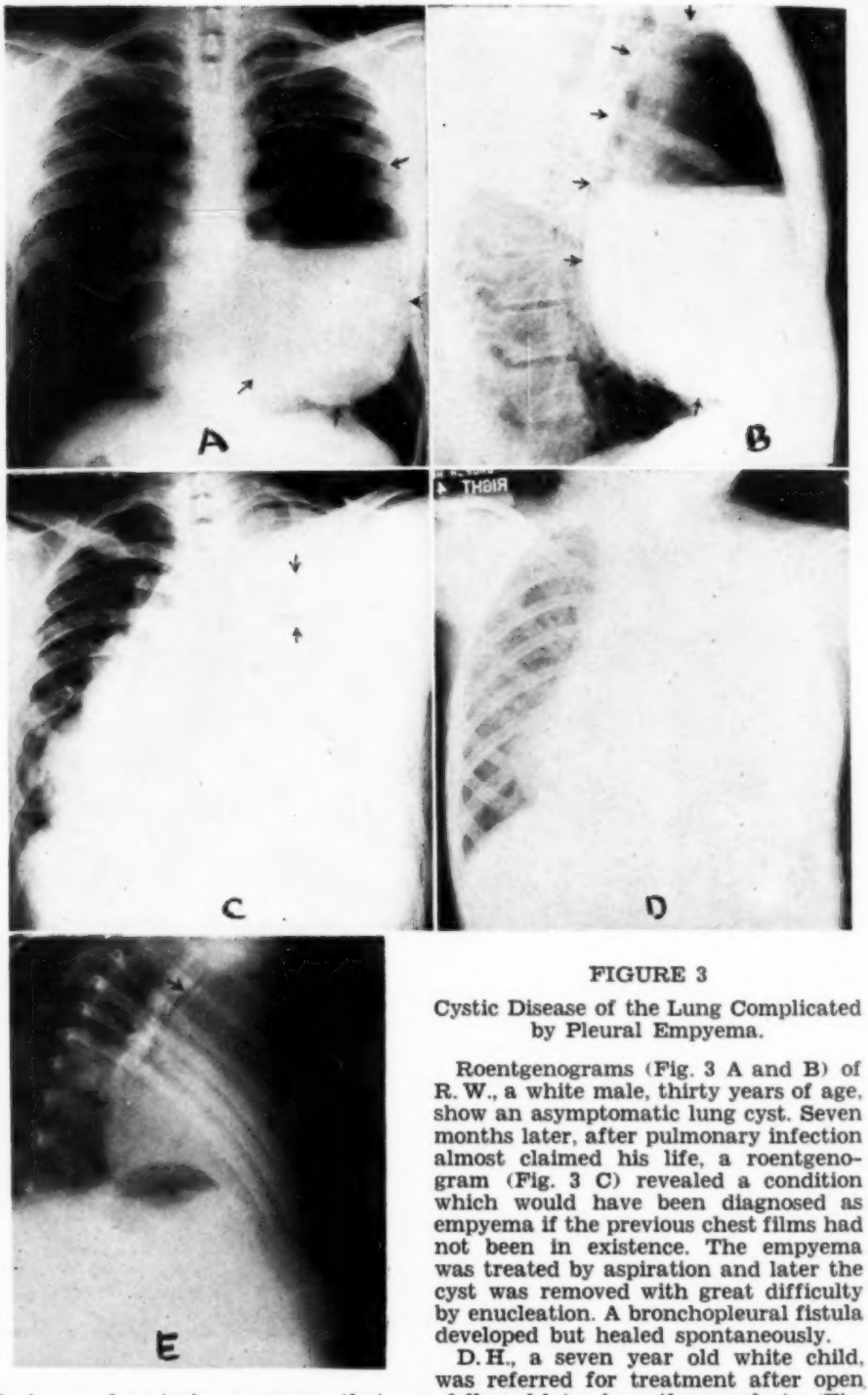

FIGURE 3

Cystic Disease of the Lung Complicated by Pleural Empyema.

Roentgenograms (Fig. 3 A and B) of R. W., a white male, thirty years of age, show an asymptomatic lung cyst. Seven months later, after pulmonary infection almost claimed his life, a roentgenogram (Fig. 3 C) revealed a condition which would have been diagnosed as empyema if the previous chest films had not been in existence. The empyema was treated by aspiration and later the cyst was removed with great difficulty by enucleation. A bronchopleural fistula developed but healed spontaneously.

D. H., a seven year old white child, was referred for treatment after open drainage of posterior empyema that was followed later by a thoracoplasty (Fig. 3 D and E). The child was still extremely ill. The similarity of symptoms to those of R. W. led to the diagnosis of pulmonary cyst, formerly complicated by empyema. Open drainage anteriorly revealed characteristics of a lung cyst. Failure to heal was predicted but fortunately there was complete recovery.

lung before operation (Fig. 6a and 6b). This patient was found to have a large systemic artery[14,22] supplying the cyst. The remaining patients had uninfected (Fig. 6) asymptomatic cysts, two of which were diagnosed during a period of severe pulmonary infection, probably secondary to the cysts. The patient corresponding to Caffey's report required no specific treatment since the cyst disappeared spontaneously. In two patients the cysts were healed completely by external drainage only. Three were extirpated by enucleation. One patient had a pneumonectomy because of bronchiectasis in the upper lobe whereas the cysts were in the lower lobe. The remainder, or eight patients, were subjected to lobectomy. There were no deaths, all have recovered completely and are asymptomatic.

By a study of these patients certain conclusions seem evident. Cystic disease of the lung is a rather common disease and, whether congenital or acquired, solitary or associated with some other disease, there is one common bond: all have an epithelial lining. Whether the cyst-like structure is congenital or acquired in origin as it develops from a minute size, it is always lined with a normal, healthy covering of epithelium. Thus, there is no break in tissue continuity; the tissues are healthy and normal. There is only a structural deformity. It is important to realize that tissue destruction by infection does not initiate the development of the cystic condition, except in the externally drained pyogenic lung abscess. If the origin of the cyst is closely associated with a bronchus it is lined with epithelium and there may be mucus glands or goblet cells. In the event these glands or cells are present the cyst will have a fluid content unless emptied by a bronchus. If the cyst arises in the manner of an emphysematous bulla it is associated with the alveoli whose walls are lined with flattened nucleated squamous epithelium.[23] Therefore the cyst will be lined with flattened epithelium that is difficult to differentiate from mesothelium. Accordingly, it will contain no mucus glands and will be air filled. The cysts, moreover, will produce no symptoms until they become infected or develop abnormal intracystic pressure, either from air or fluid.

Many times it has been reported that a lung abscess may become epithelial lined by the ingrowth of bronchial epithelium from communicating bronchi. Incontestable proof of such an assumption has not been found by the author. It must be accepted that such a condition might occur but only under ideal circumstances. First, it would be necessary to have complete elimination of the infection and, accordingly, the cavity would obliterate before epithelization could occur. Increased intracavitary pressure could prevent collapse of the cavity while epithelization takes place.

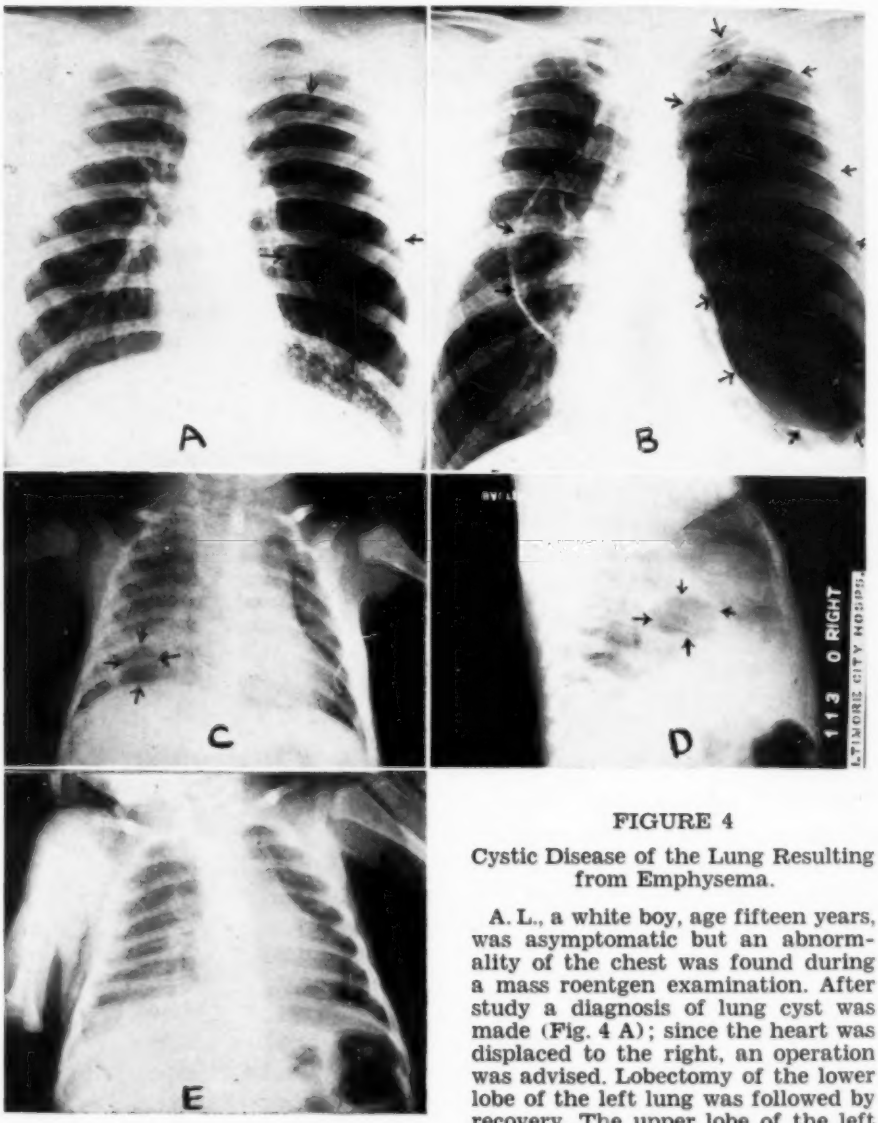

FIGURE 4

Cystic Disease of the Lung Resulting from Emphysema.

A. L., a white boy, age fifteen years, was asymptomatic but an abnormality of the chest was found during a mass roentgen examination. After study a diagnosis of lung cyst was made (Fig. 4 A); since the heart was displaced to the right, an operation was advised. Lobectomy of the lower lobe of the left lung was followed by recovery. The upper lobe of the left was normal but the lower lobe showed considerable emphysema without a definite cyst.

C. J. was a white man, age forty-one years, with a gradual but progressive increase in dyspnea over a four year period. A diagnosis of spontaneous pneumothorax was made thirty months ago. During the past thirty months the cyst became enlarged by herniation into the right chest through the mediastinum (Fig. 4 B). At operation the cyst wall was easily freed and was found to originate from the upper part of the upper lobe of the left lung. Several smaller areas of emphysematous bullae or cysts were present in the adjacent area. Excision of the cyst with the adjoining emphysematous bullae required the removal of only a small portion of the lung. The remainder of the lung appeared normal and expanded rapidly. The patient made an uneventful recovery.

R. S., a white male, age two months, developed a pulmonary infection. A chest roentgenogram taken January 15, 1945 (Fig. 4 C and D) is shown. The infant made a satisfactory recovery on appropriate medication and a roentgenogram taken January 31, 1945 (Fig. 4 E) revealed no evidence of the cavity or cyst. The last roentgenogram of the chest was taken March 14, 1945 and the cavity had not

Such pressure would indicate incomplete drainage; therefore, infection could not be overcome and epithelization could not take place. In considering the incidence of pyogenic lung abscess there would be a great many more epithelial lined spaces encountered if epithelization were actually a step in the pathogenesis of pyogenic lung abscess. Many authors have opposed this point of view. [9,10,24] The lattice lung described as an epithelized lung abscess is strikingly similar to the one described as an infected lung cyst.[9,24,25] Thus the question arises as to whether they may not be one and the same condition. Patients with infected pul-

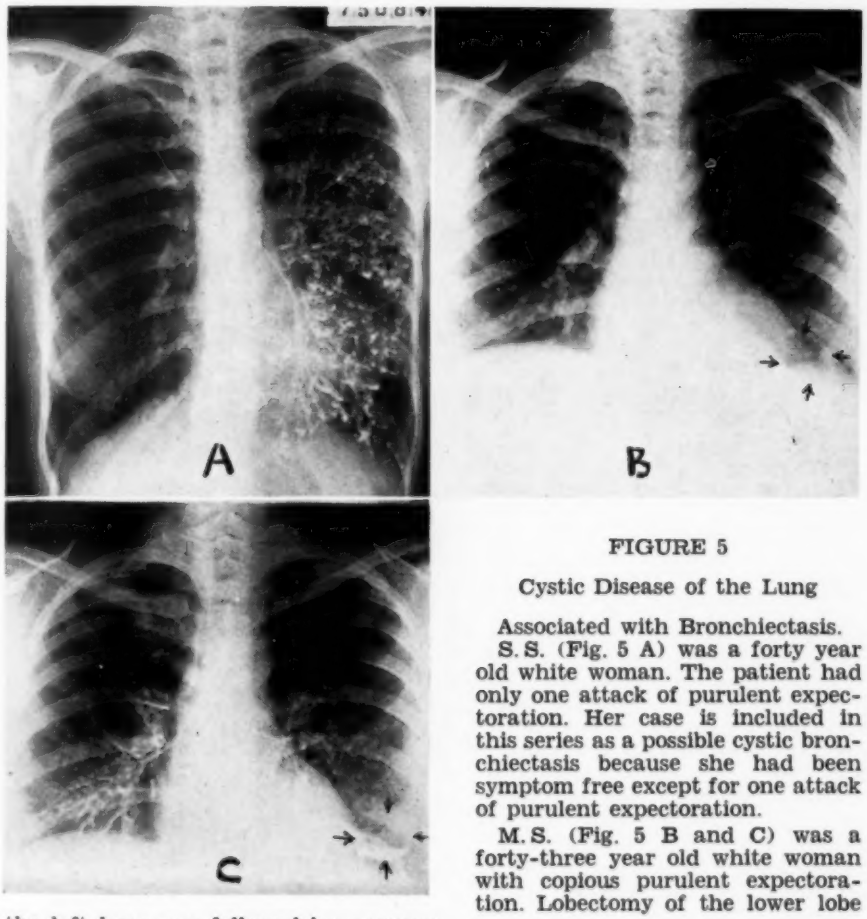

FIGURE 5

Cystic Disease of the Lung

Associated with Bronchiectasis.

S. S. (Fig. 5 A) was a forty year old white woman. The patient had only one attack of purulent expectoration. Her case is included in this series as a possible cystic bronchiectasis because she had been symptom free except for one attack of purulent expectoration.

M. S. (Fig. 5 B and C) was a forty-three year old white woman with copious purulent expectoration. Lobectomy of the lower lobe of the left lung was followed by recovery.

M. S. (Fig. 5 B and C) and M. G. (Fig. 5 C, D and E) are probably patients with bronchiectasis secondary to or merely associated with pulmonary cysts.

FIGURE 4 — Continued:

reappeared. This patient represents the condition termed regional obstructive emphysema described by Caffey.

It appears likely that this group of cysts was emphysematous in origin. A. L. did not have a lung cyst but his roentgenogram is shown since perhaps it is a stage in the development of an emphysematous type of lung cyst.

monary cysts diagnosed as lung abscess have a mild course when compared with the course of a true pyogenic lung abscess, and the chest roentgenograms fail to reveal retraction of the chest and thickened pleura. Of course, when the infected lung cyst is drained it usually will not heal because of the epithelial lining. The origin of pyogenic lung abscess by suppurative tissue destruction would definitely set it apart from an infected pulmonary cyst.

If a pyogenic abscess of the lung is drained externally a condi-

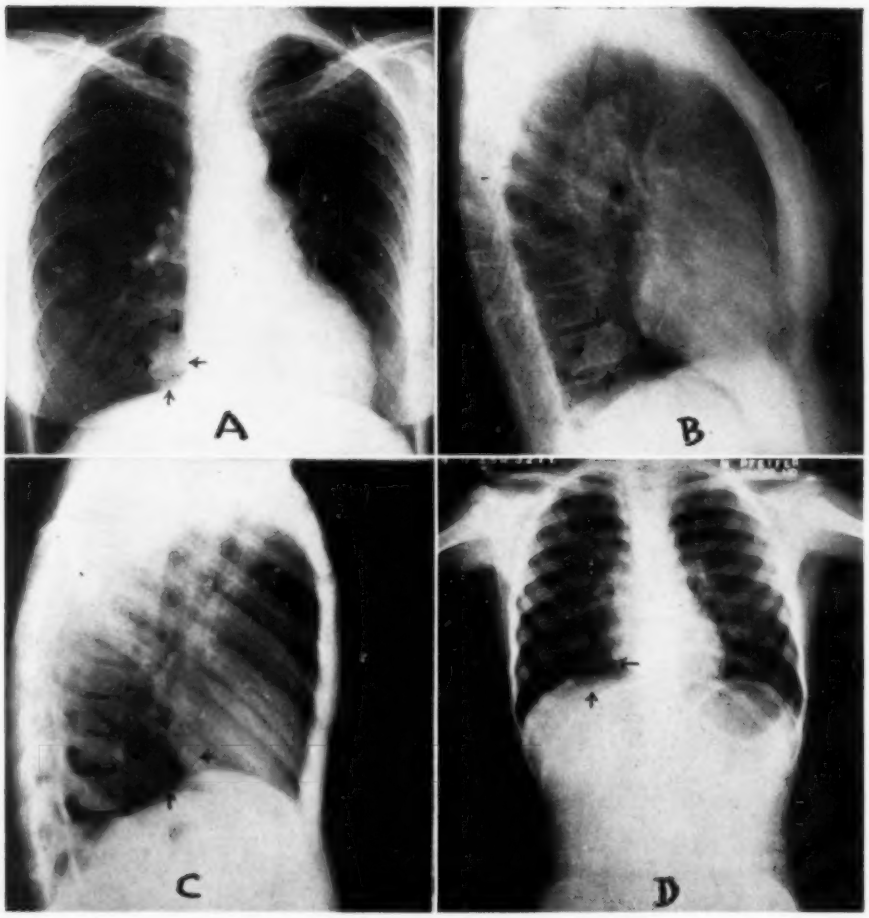

FIGURE 6: Cystic Disease of the Lung, Asymptomatic and Uncomplicated.

M. A. (Fig. 6 A and B) was a fifty-six year old white woman who expectorated blood on only one occasion. Carcinoma of the lung was the preoperative diagnosis. Lobectomy of the lower lobe of the right lung was followed by recovery. A large, pencil size systemic artery entered the lung at the region of the cyst.

N. P. (Fig. 6 C and D) a five year old white girl ,had no symptoms. Lobectomy of the lower lobe of the right lung was followed by recovery. It is interesting to note that the cyst is the third of the series that contained only air. This cyst was within the lung parenchyma instead of on the surface as represented by C. J. (Fig. 4 B). The cyst in this patient first was found at the time of a pulmonary infection, but after many months' observation it did not disappear as in R. S. (Fig. 4 C, D and E).

tion suitable for epithelization is produced. The infection is overcome and, if for any reason the space does not collapse, epithelium from the communicating bronchi can grow into and cover the walls of the space. This view is held by Edwards.[25] The epithelized space continues to drain because of the presence of mucus glands and goblet cells in the lining membrane. Bronchial communication is usually free and if the external wound is allowed to heal there results the equivalent of an epithelized cyst. When more

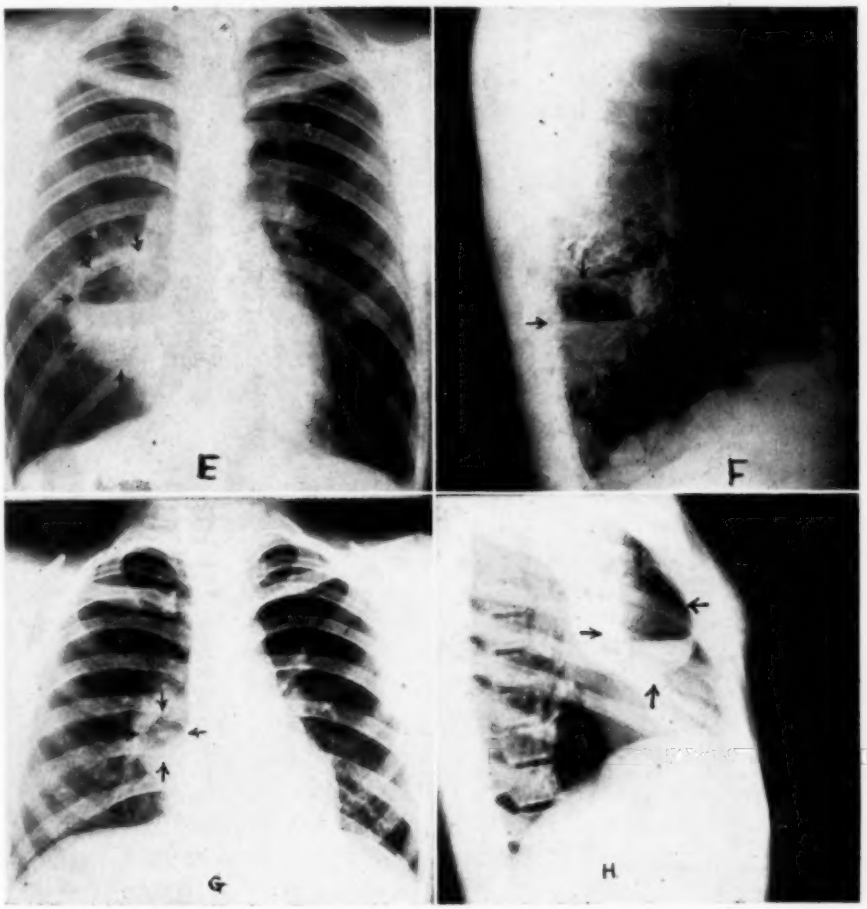

FIGURE 6 (Continued): Cystic Disease of the Lung, Asymptomatic and Uncomplicated.

J. R. (Fig. 6 E and F) was a twenty-two year old white man who had a roentgenogram of the chest following a pulmonary infection three months before. He remained free of symptoms. Lobectomy of the lower lobe of the right lung was followed by recovery.

J. W. (Fig. 6 H and G) a thirteen year old white girl, had a course similar to J. R. The cyst was enucleated with recovery.

These four patients represented three types of cysts, one completely filled with fluid, one completely filled with air, and two with both air and fluid. These cysts are probably congenital in origin.

than one bronchus communicates with an epithelized space it seems reasonable to expect that the space is an old epithelized lung abscess. Two of the cases (Fig. 1a, 1b and Fig. 1c, 1d, 1e) herein reported undoubtedly represent epithelized cavities occurring after drainage of a lung abscess. Both patients presented trabeculation within the cavity and both had many bronchi leading into the epithelized cavity. The history of external drainage shortly after the onset of the disease sets them apart as instances of epithelized lung abscess.

The healing of tuberculous cavities by epithelization has been described.[26-28] If healing by epithelization were a step in the pathogenesis of tuberculosis there would be on record many examples, since tuberculosis with cavitation is a common disease. One can assume that such cases as are described might easily have been lung cysts and that pulmonary tuberculosis was coincidental. Many cases of cystic disease of the lung have been diagnosed incorrectly as tuberculosis.[1,3]

Since the pulmonary cysts in cystic lung disease are not initiated by infectious tissue destruction, the onset is insidious and silent. From their inception the cysts are lined with a healthy epithelium. Therefore, they are resistant to infection because there is no portal of entry. The usual absence of pleural infection when air escapes from healthy parenchyma of the lung into the pleural cavity would indicate that regardless of the type of bacteria present in the upper respiratory passages, the lower portions of the lung are normally free of bacteria. The pulmonary cysts are generally connected with bronchioles or respiratory bronchioles, or even alveoli, and therefore, they are seldom exposed to infection. When infection does occur it is usually rather mild because of the resistance and limiting effect of the lining membrane.

It is difficult to associate bronchiectasis with cystic disease of the lung. However, a cystic type of bronchiectasis does occur and sometimes the cysts are large.[3] It can be assumed that epithelial lined dilitations occur without tissue destruction by infection and often remain asymptomatic. Because of the lining membrane the course of an infected cystic bronchiectasis is mild compared to bronchiectasis that develops by actual tissue destruction. One patient in the series (Fig. 5a) probably represents cystic bronchiectasis. Her only symptoms occurred during a short period of secondary infection. The patients shown in Fig. 5b, 5c and Fig. 1c, 1d, 1e probably represent bronchiectasis associated with or secondary to cystic disease of the lung.

The mechanism of the development of the cyst-like structures from emphysematous bullae and the mechanism for development of subsequent pressure symptoms have been described adequate-

ly.[29,30] It is important to point out that the condition occurs in adults as well as in infants or children.

In the two patients who recovered completely after simple open drainage it can be argued that they did not have infected lung cysts. However, both had epithelial lined cavities and typical trabeculations much like the lattice lung. Neither patient had a previous external drainage. One can assume that destruction of the lining membrane was so complete that healing ensued. Failure to heal was predicted for both.

In a discussion of cystic disease of the lung, the cysts of the pleura and mediastinum should be excluded and most authors also omit the echinococcus cysts.[2,31] The classification of Dickson, Clagget and MacDonald[11] with minor modifications seem highly satisfactory.

I. The true developmental or so-called congenital cysts of the lungs (some of the bronchogenic cysts or bronchoalveolar cysts).

II. Acquired cysts or cyst-like cavities of the lungs.

  A. Cystic bronchiectasis

  B. Other pulmonary cysts or cyst-like cavities

    1. Emphysematous bullae
    2. Obstructive emphysematous cyst of Caffey
    3. Abscess cysts (after external drainage)
    4. All other pulmonary cysts.

The obstructive emphysematous cyst described by Caffey and represented by Fig. 4c, 4d and 4e should have a prominent place in any classification since the condition is distinctive. It occurs only in infants and it regresses to complete obliteration on appropriate medication for pulmonary infection. Spontaneous obliteration has not occurred in infants over one year of age.

Extirpation of the cyst is the treatment of choice. The method of extirpation depends upon the location of the cyst. If it is placed peripherally to the lung parenchyma, it can be enucleated or removed by partial lobectomy. If the cyst is completely surrounded by lung parenchyma, lobectomy is the procedure of choice. The only pneumonectomy performed in the present series was made necessary by the cysts involving the lower lobe of the lung and the upper lobe of the lung was involved extensively by bronchiectasis. Severe infection may make preliminary drainage of the cyst imperative. When open drainage of the pleural cavity or pulmonary suppurative condition is accomplished a biopsy should be made. It may lead to the correct diagnosis of the condition.

CONCLUSIONS

1) An epithelial lining membrane is the common characteristic that binds together the apparently diversified conditions included under the term "Cystic disease of the lung."

2) As a result of the epithelial lining membrane the pulmonary cyst, whether congenital or acquired, presents definite symptom patterns, and runs a mild course when infected.

3) Because pulmonary cysts have an epithelial lining membrane, extirpation is the treatment of choice.

CONCLUSIONES

1) La característica común a todos los estados aparentemente diversos que se incluyen bajo el término de "Enfermedad Quística del Pulmón" es que tienen una membrana epitelial.

2) Por razón de la membrana epitelial el quiste pulmonar, ya sea congénito o adquirido, presenta complejos sintomáticos bien definidos, y evoluciona levemente cuando está infectado.

3) Debido a que los quistes pulmonares tienen una membrana epitelial, la extirpación quirúrgica es el tratamiento de elección.

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Discussion

L. M. SERRA, M.D.
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Dr. Brantigan is to be congratulated for his excellent paper which represents an exhaustive analysis of numerous articles which have appeared particularly during the past few years; and is the result of a large and successful experience in the surgical management of cystic disease of the lung, which permits certain definitive conclusions. As an internist, there is little that I can add; indeed, my few cases were treated surgically by him. One, a simple asymptomatic cyst which was discovered accidentally. Another, also discovered accidentally, proved to be a large emphysematous area. At the time of operation, this patient presented something of a problem regarding what to do. Dr. Brantigan decided to remove the involved lobe, and the heart, which had been somewhat displaced, returned to normal position. After operation, the case was considered carefully, and it was decided, particularly by Dr. Brantigan, that in similar instance, surgical removal would be indicated. The third case we both felt was an

acute lung abscess; at operation it was found to be an infected cyst, which was appropriately drained with the idea of follow-up surgery at a later date. Interestingly enough, the wound has completely healed, the man is perfectly well, and his last roentgenogram is negative.

While cystic disease of the lung is by no means rare, one may wonder whether we are justified in referring to it as "common." Dr. Brantigan has certainly seen a large number during the past seven years; however, we must realize that these cases are, to use his words, channeled to him as a thoracic surgeon practicing in a large city.

As regards diagnosis, this does not present too much difficulty as a rule; however, there are instances, as noted previously, in which the true nature of the process may be missed. If full of fluid, the matter of a tumor, let alone abscess, empyema, aneurysm and dermoid cyst, must be ruled out.

Dr. Brantigan reports one instance in which an unusual vessel was encountered; this calls to mind that cystic disease of the lung has not infrequently been associated with various pulmonary anomalies, such as accessory lobes and aberrant vessels; the latter should particularly be kept in mind at operation.

The rapid strides in thoracic surgery during the past ten years have made the surgical removal, by one or the other of the methods enumerated by Dr. Brantigan, an almost universally accepted procedure. The patients presenting symptoms as a result of cystic disease, either simple or complicated, certainly should be subjected to surgery, unless some particular contraindication be present; these need no comment. It has been repeatedly stated that uncomplicated cystic pulmonary lesions must be uncommon; this in a measure is substantiated by the infrequency of the lesion at the autopsy table, and in the course of the mass x-ray surveys being conducted throughout the country. One cannot help but be impressed with the relative benignity of the complicated cystic disease; this is amply demonstrated by Dr. Brantigan's cases and those appearing in the literature. Realizing the infrequency of the simple cystic disease without symptoms, there is probably no point in arguing against the advisability of surgery in these instances. In culling the literature, one is impressed by the fact that most articles have been written by surgeons or roentgenologists. With these facts in mind, would it be too far off the beaten path to suggest expectant treatment, with frequent roentgen studies, in the uncomplicated, asymptomatic cases in which the cyst is not too large.

Dr. Hudson called attention to the possibility of malignancy developing in cysts of the lung. I had not been impressed by this

possibility and, if proven, would, of course, preclude any expectant therapy.

Cystic disease of the lung, first recognized about two hundred and fifty years ago, made its real debut as a supposedly congenital condition in Dr. Koontz's paper in 1925. As alluded to in Dr. Brantigan's paper, the pendulum has swung well toward the side of the condition often being acquired. The disease has now come of age; it has achieved an important place in chest diseases and deserves our careful consideration.

Discussion

WILLIAM A. HUDSON, M.D.
Detroit, Michigan

Dr. Brantigan very wisely reviews the different theories concerning the origin of pulmonary cysts. It would seem proper to use the term Cystic Disease of the Lungs to include all cyst-like conditions which cannot be definitely associated with some indisputable cause or origin until such a time as we shall find some indisputable evidence by which we can differentiate the acquired from the congenital cysts of the lung.

He wisely points out the value to clinical history and clinical findings when considered together with proper laboratory procedures in the diagnosis of such conditions. The treatment is, for the most part, surgical.

I reported a case of acquired cyst of the lung at the Meeting of the Southern Chapter, of the American College of Chest Physicians in 1946, showing photographic records of the cyst with the inner wall of the cyst being formed by denuded lung; the outer wall formed by visceral pleura. This cyst-like structure had apparently been produced as a result of a sudden compression of the chest in an automobile accident several months previously. There were x-ray films available to support this contention.

May I congratulate Doctor Brantigan on his fine presentation.

The Problem of Bronchiectasis*
A Review

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North Hollywood, California

Bronchiectasis, like pulmonary tuberculosis, is a killer of men; but bronchiectasis, unlike tuberculosis, kills on a fairly definite schedule. Tuberculosis, unless in a terminal state, tends to heal if given a chance; and we see many people who once had far advanced active pulmonary tuberculosis, who have regained a measure of health and economic independence; but we see very few bronchiectatic patients with far advanced disease who are ever able to do more than live a miserable, dependent existance; the tendency of bronchiectasis is to progress downward. Head[1] found, in a group of two hundred bronchiectatic patients, who had acquired their disease in the first ten years of life, that few were living after forty years of age. Riggins,[2] from observations at the Bellevue and Lennox Hill Hospitals, New York City, agreed with Head for the most part; but also saw a fair sprinkling of bronchiectatic patients in their sixth or seventh decades. While Roles and Todd[3] observed a forty-seven per cent mortality in forty-nine non-surgically treated patients that were followed for six years. Clagett and Deterling, Jr.,[4] stated that some bronchiectasis was found in two per cent of all the necropsied cases at the Mayo Clinic.

The bronchiectasis found in the right upper lobe and in the upper portion of the left upper lobe due to atelectasis, bronchial stenosis, and contracting scar tissue and following various inflammatory conditions of the lung (including tuberculosis), is relatively benign because the drainage is downhill; the drainage of bronchiectatic lesions in the lower lobes, right middle lobe, and lingula of the left upper lobe is uphill, and more liable to pursue a progressive course. Also, there may be a more or less temporary bronchiectasis following acute respiratory infections as bronchitis, virus pneumonia, and which follows enlarged hilar glands as from primary phase tuberculosis; in these cases the bronchiectasis follows bronchial stenosis with its tendency to trap inspired air and prevent its egress, combined with areas of atelectasis which increase the negative pull of the pleural space; this type of bronchiectasis has a tendency toward recovery when the

*Read before the Glendale Branch of The Los Angeles County Medical Association, at Glendale, California, October 20, 1947.

causative conditions are removed, provided the bronchial walls do not become grossly infected.

Bronchiectasis, as we ordinarily understand it, is an acquired dilatation of the bronchi and bronchioles and the primary lobules or lung units of the pulmonary parenchyma. But before going further into the etiology, pathogenesis, pathology, and treatment of bronchiectasis, it seems advisable to briefly review the developmental and final anatomy of the tracheo-bronchial tree and lungs in order that these phases may be better understood.

The anlage of the larynx, trachea, bronchi, and lungs arises about the fifteenth day (embryo of 23 segments; 3.2 mm. in length) from an outgrowth in the ventral wall of the entodermal tube (the foregut; the primitive oesophagus). This anlage is known as the laryngotracheal groove. The caudal end of this outgrowth promptly becomes rounded and marks the future development of the bronchi and lungs; constriction of the groove proceeds cephalad and it becomes separated from the primitive oesophagus, the cephalic portion becoming the larynx and the intermediate part becoming the trachea[5,6] The rounded, caudad portion, the lung bud, becomes bilobed in four to five millimeter embryos. Both lung buds elongate, the right being slightly larger than the left at first. These lung buds (stem buds, main bronchial stems) grow downward, lateralward and backward, branching monopodially to give rise to the future bronchi. In the later stages of development there is probably dichotomous branching of the smaller bronchi.[5,6]

In the seven millimeter embryo, from the stem bronchi, two bronchial buds arise on the right side (the apical bud, future right upper lobe bronchus; and a ventral bud, the future right middle lobe bronchus); a ventral bud also arises on the left bronchus (the future left upper lobe bronchus); the main bronchial stems or buds become the lower lobe bronchi.[5,6] These future bronchi grow into a mass of mesenchymal cells and carrying before them folds of mesoderm. The mesenchyme forms the cartilage plates, muscle, connective tissue of the lungs, and tracheal and bronchial walls. Blood vessels and nerves grow into this developing mass. The visceral (splanchnic) mesoderm forms the visceral pleura, and the somatic (parietal) mesoderm forms the parietal pleura. The entoderm of the primitive trachea and bronchi forms the epithelium lining of these and the lining of the lung units.[5,6] The air cells or alveoli begin to form at about the sixth month; and the lung is fully formed when birth takes place.[5,6]

Macklin[7] considers the tracheo-bronchial tree as far as the respiratory bronchiole, as merely a conduit system; while the

part beginning with the respiratory bronchiole and ending with the pulmonary alveoli is the functional part. The bronchi, when they enter the lungs, become cylindrical and gradually acquire a circular layer of smooth muscle on the outer border of the tunica propria. These muscle fibers become more and more prominent as the smaller bronchi are reached, and are found to extend as far as the atria.[6] The other elements of the bronchial wall (cartilage, white fibrous tissue, elastic tissue, glands, etc.) decrease as the smaller bronchi are reached. The circular muscle is best developed in the terminal bronchiole, and can exert a strong sphincteric action in bronchospasm;[8] as the bronchi grow smaller, the cartilagenous rings change to small plates or flakes, and disappear in bronchi of about one millimeter diameter.[6] The mucus glands also disappear about here[6] (Fig. 1).

Stratified, ciliated, columnar epithelium lines the trachea and larger bronchi; as the bronchi grow smaller, this becomes simple, columnar, ciliated epithelium; then ciliated cuboidal epithelium; when the respiratory bronchiole is reached, the epithelium becomes simple cuboidal with areas of squamous or respiratory epithelium. The remaining portion of the tract (alveoli, air cells) are lined with squamous epithelium; probably deficient[8] in places (Fig. 2).

FIG. I

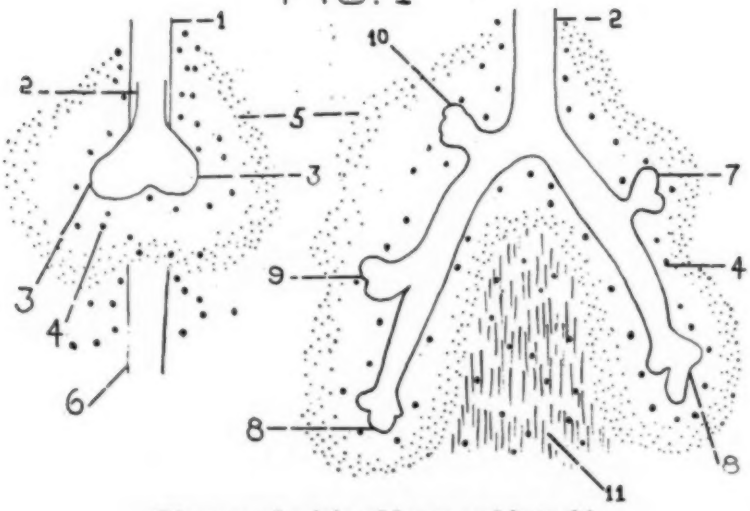

Diagram of origin of lungs and bronchi.

1, Entodermal tube (fore-gut); 2, primitive trachea; 3, lung bud from ventral aspect of entodermal tube; 4, mesenchyme; 5, mesothelium (visceral), future visceral pleura; 6, primitive esophagus from fore-gut; 7, ventral bud (future left upper lobe bronchus); 8, stem buds (main bronchial stems); 9, ventral bud (future middel lobe bronchus); 10, apical bud (future right upper lobe bronchus). On left side, apical bud in ventral bud; no separate lobe on left side comparable to right upper lobe; 11, septum transversum (future diaphragm).

Miller's[12] lung unit begins with the respiratory bronchiole (bronchiolus respiratorius) which gives off several alveolar ducts (ductili alveolaris; alveolar passages; vestibules); each alveolar duct gives rise to several irregularly spherical dilated parts, the atria; each atrium originates several alveolar sacs (sacculi alveolaris; infundibula; air sacs); and in each alveolar sac wall, there arise a number of alveoli (alveoli pulmonis; air cells). Best and Taylor's[8] description of a lung unit or primary lobule begins with the respiratory bronchiole, which is a continuation of the terminal bronchiole, and has the same length (0.2 - 0.5 mm.) and diameter (0.3 - 0.4 mm.); five or six alveolar ducts arise from one respiratory bronchiole; and an alveolar duct gives origin to three to six alveolar sacs after a variable number of rebranchings; an alveolar sac contains a number of small pouches, the alveoli. As the bronchioles approach the periphery of the lung, they grow shorter, but maintain about the same diameter as the earlier ones (0.3 - 0.4 mm.), the first branches being about 1.5 millimeters in length.[8]

FIG. 2.

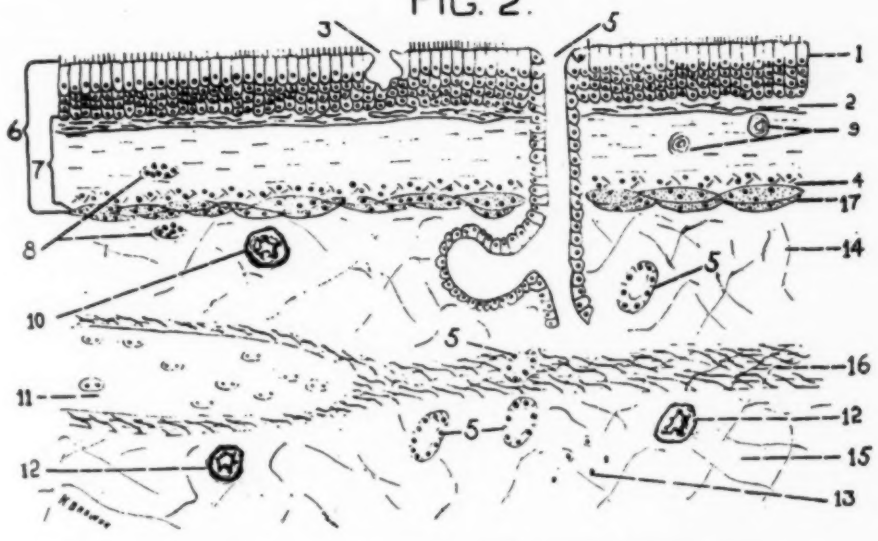

Diagram of cross section of medium-sized, intrapulmonary bronchus.

1, Stratified, ciliated, columnar epithelium; 2, basement membrane (membrana propria); 3, goblet, mucus cell; 4, outer layer of tunica propria, shows longitudinal elastic fibers cut across, and white fibrous tissue, inner layer shows fine, loose fibers and basement membrane; 5, racemose mucus and serous gland opening on mucus surface and extending into adventitia; 6, mucus membrane; 7, tunica propria (corium); 8, lymph gland; 9, 10, 12, blood vessels. All layers contain blood vessels, nerves, lymphatics and secreting glands; 11, hyaline cartilage plate in dense fibro-elastic layer; 13, fat cells; 14, submucosa of coarse, loose areolar tissue with glands, vessels, nerves and lymphatics; 15, adventia of loose areolar tissue with glands, vessels, nerves and lymphatics; 16, fibro-elastic layer of white fibrous tissue and yellow elastic fibers densely arranged; 17, circular, smooth muscle layer in mucus membrane.

The essential etiology and pathogenesis of bronchiectasis can be summed up in a few words: Stenosis of a bronchus with retained infective secretions permit infection of the bronchial walls, with weakening of such stromal elements as elastic tissue and muscle, and permit the rythmical pull of inspiration, possibly combined with the weight of the pooled secretions, gradually to dilate the walls of the bronchi and lung units into permanent or potential cavities. When there is atelectasis of portions of the surrounding lung; or when there is contracting, inelastic, fibrous tissue in the surrounding lung, this inspiratory pull is greatly increased; when there are also partly occluded bronchi which permit air to enter, but which exert Chevalier Jackson's well known check-valve action in preventing egress of some of the air, the dilating effect on the infection-weakened walls is heightened.

The bronchiectasis, mainly in the upper lobes, caused by the irregularly developing fibrosis of chronic tuberculosis produces dilatation in the distorted, gnarled bronchi which does not present the regular pattern seen in ordinary lower lobe bronchiectasis with its more evenly developing fibrosis.[13] Even so, this upper lobe bronchiectasis of tuberculous fibrosis is usually relatively benign because the drainage is downhill; but the bronchiectasis that follows invasion of the bronchial walls by tubercle bacilli, with possible secondary infection by ordinary pyogenic bacteria, is one of the severer complications of pulmonary tuberculosis, and may require surgical removal.

Bronchiectasis can develop at any age; but a large percentage of cases develop during the first ten years of life. Perry and King[14] claimed that the onset of 42 per cent of their patients was during the first ten years, and that the onset occurred in 27 per cent during the second ten years. Farrell[15] stated that 80 per cent of his bronchiectatic patients acquired their disease during the first ten years.

Singer and Graham[16] pointed out that the dense, triangular shadows seen along the mediastinal borders with bases on the diaphragmatic leaflets, apices in the hili, and hypotenuses facing peripherally were due to atelectasis of lower lobes. These are seen principally in infants and young children. There are similar, but larger and less dense triangular shadows which are due to interstitial infiltration and fibrosis in bronchiectasis.

McNeil, MacGregor and Alexander,[17] Richards,[18] and Anspach,[19] showed that these triangular areas of atelectasis frequently accompanying pneumonia, were often followed by bronchiectasis later, especially in infants and children. The smaller bronchi of infants and children are especially susceptible to plugging, from thick, tenacious secretions with attendant atelectasis. It is a law

of the cube that the smaller bronchi would present a relatively greater surface for adherence per unit of volume than the larger bronchi.

Anything which causes bronchial stenosis predisposes to bronchiectasis. The inflamed bronchi of whooping cough, measles, bronchopneumonia, and bronchitis, are special sources in childhood; enlarged tracheobronchial and hilar glands following acute and chronic infections, and tuberculous adenitis of these glands may cause stenosis of the bronchi with development of bronchiectasis. The inhalation of foreign bodies with the drowned lung beyond the obstruction are frequent sources of bronchiectasis, particularly in children; and as Chevalier Jackson pointed out long ago, the vegetable body as the peanut is more liable to encourage bronchial and pulmonary suppuration than the metallic one.

There is a form of chronic bronchitis and bronchiectasis occurring in children having cystic fibrosis of the pancreas. The lack of exocrine secretion of the pancreas with failure to digest fats and other nutritional constituents causes lack of absorption of vitamines A and D and malnutrition; these in turn permit infection of the respiratory tract with pyogenic organisms, espec-

FIG.3

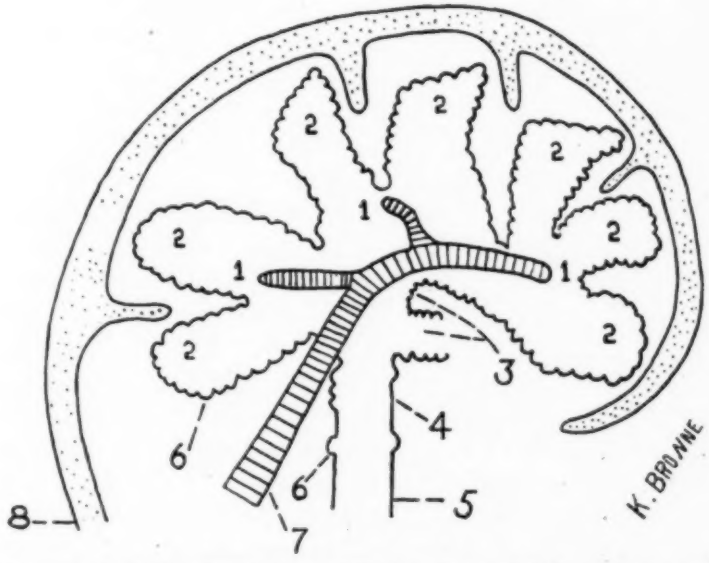

Diagram depicting William S. Miller's primary lobule or lung unit.

1, atria; 2, alveolar sacs (infundibuli); 3, alveolar ducts (vestibuli); 4, respiratory bronchiole; 5, terminal bronchiole; 6, alveoli (air-cells); 7, pulmonary arteriole accompanying terminal bronchiole, and giving a branch to each atrium; then forming capillary plexuses around alveoli; 8, pulmonary venule collecting from plexuses; then following partly independent course back to heart, but joining other venules.

ially staphylococci; as with other vitamine A deficiencies, there is a piling up of epithelial debris in the salivary gland ducts, trachea and bronchi.

Kartagener[20] found bronchiectasis and sinusitis frequently associated with dextrocardia and situs inversus, the "Kartagener triad." He cited this in support of the theory that bronchiectasis is congenital. Adams and Churchill[21] reported five cases of this syndrome. Olsen[22] reviewed 85 cases of dextrocardia seen at the Mayo Clinic from 1920 to 1941, inclusive; evidence of bronchiectasis was found in 16.5 per cent of these cases in contrast to less than 1 per cent of bronchiectasis in all other patients registered there. He also mentioned bronchiectasis occurring in pairs of identical twins as further evidence for the congenital theory of bronchiectasis.

Any condition of the lungs and bronchi that causes fibrosis or bronchial stenosis or both as pneumonoconiosis, corrosive gases, fungus infections, bronchopneumonia, bronchitis, pleural empyema, lung abscess and the like, may be causative of bronchiectasis. Benign or malignant intrabronchial tumors; extrabronchial tumors; large glands, or fibrotic bands pressing on the bronchial walls all encourage the development of bronchiectasis. Uncomplicated pneumococcus lobar pneumonia rarely causes bronchiectasis, because the involvement here is primarily parenchymal and not bronchial. But the virus types of pneumonia are frequently followed by the development of bronchiectasis. Here, there is an intense inflamatory involvement of the bronchial and peribronchial tissues as well as an alveolar exudate and hemorrhagic pneumonitis. The partly occluded bronchi with their damaged walls and retained infective secretions, the scattered areas of atelectasis all favor the invasion of secondary bacterial pathogens into the bronchial walls and surrounding tissues.

Watson and Kibler[23] reiterated an earlier theory of theirs that allergy of the bronchial tubes with its attendant bronchial stenosis and retention of secretions was the most frequent cause of bronchiectasis. It is true that the bronchial swelling and muscle spasm of allergy furnish a good foundation for the development of bronchiectasis; but is not the only mechanism that does so.

The pathological changes in bronchiectasis are varied. The invasion of the bronchial walls and surrounding lung tissue by the pathogenic bacteria and defence white cells of the blood is followed by varying degrees of destruction and replacement by inelastic, white fibrous scar tissue. The scar tissue, when young and gelatinous, is easily stretched by inspiration and the weight of retained secretions; but becomes hard and contracted when older. There is loss of the elastic elements in the bronchial walls.

The ciliated, columnar epithelium may be eroded in places, leaving granulation or scar tissue in its place; or it may be replaced by flattened epithelium devoid of a ciliary fringe with its self cleaning action. Blocked, infected secretions may extend into the lung, producing lung abscess or pleural empyema.

After each bout with an acute respiratory infection, the chronic bacterial infection spreads more extensively into the bronchial walls and lung parenchyma, leaving more destruction and scar tissue formation. A greater and greater load is added to the right side of the heart by the mechanical obstruction to blood flow and the replacement of functional units in the lung. This recurs until the right ventricle fails and eventually congestive heart failure develops. This heart failure responds to cardiac treatment for a time; but if the patient does not die subsequently of metastatic abscess, pneumonia amyloid disease, nephritis, or massive hemorrhage, he will eventually die of heart failure from the chronic overwork, and infectious or toxic myocarditis from the pulmonary infection.

The left lower lobe is the one most frequently involved in bronchiectasis; and when bronchiectasis develops here, the lingular segment of the left upper lobe frequently becomes affected. Churchill and Belsey[24] found the lingular segment sufficiently involved to require surgical removal in eighty per cent of cases requiring removal of the left lower lobe. This frequent left sided localization is probably mainly a matter of drainage; the left main bronchus and its branches make more of an angle with the trachea than does the right one, which is more of a continuation of the trachea. Clagett and Deterling[4] found the following lobar distribution in 471 bronchiectatic patients seen at the Mayo Clinic: the right lower lobe in 19 per cent; the left lower lobe in 35 per cent; both lower lobes in 19 per cent; the right middle lobe seldom. While these statistics agree fairly with those of others, there are a considerable number of patients who have right middle lobe bronchiectasis accompanying bronchiectasis of the right lower lobe.

There is no micro-organism distinctive of bronchiectasis; but streptococci (including viridans or alpha type, hemolyticus or beta type, non hemolyticus or gamma type and probably some anerobic types), seem to play an especially prominent role in bronchiectasis. The pathogenic significance of streptococcus viridans is not yet settled. Monilia albicans is found occasionally as a secondary saprophitic invader, but may possibly at times assume pathogenic importance. The fusiform-spirilla group is only rarely found in bronchiectasis; and then mainly as a saprophytic invader. The bad odor found in the sputum of some bronchiectatic patients

is now thought to be rarely caused by this group. This odor is now attributed by many, to other organisms, as the anerobic streptococcus. The Neisseria catarrhalis is considered by many to play little or no part in the disease process.

In some quarters, there is a marked swing away from the former tenet that sinusitis is a cause of bronchiectasis; and some present day observers even claim that the sinusitis present is caused by the bronchiectasis. I have found that sinusitis is very frequently associated with bronchiectasis; and that very little permanent result can be secured by either medical or surgical treatment in bronchiectasis if the accompanying sinusitis cannot be controlled; and very often a case of early bronchiectasis can be improved by merely clearing up the sinusitis present. The protagonists of the sinusitis etiology of bronchiectasis postulate two possible routes of spread of infection to the bronchi: the inhalation route, and the lymphatic route, the former having the most supporters.

The diagnosis of advanced bronchiectasis is usually easy; the coughing up of large amounts of purulent sputum; the dyspnoea and cyanosis; the clubbed fingers; the low grade temperature with acute exacerbations; the history of frequent pneumonia like attacks, especially during the cold months; the hemoptysis varying from slight streaking, to massive pulmonary hemorrhage, all give definite leads to the diagnosis. There is present at some time, a myofascitis, especially along the paravertebral muscles, and often segmental neuralgia; frank rheumatoid arthritis is also seen, but less often.

To diagnose early bronchiectasis, or the prebronchiectatic state, constant diagnostic suspicion and readiness to act are necessary; what seems to be unresolved pneumonia may be an atelectatic lobe with bronchiectasis developing; the subacute sinusitis, or bronchitis, may already be complicated by early bronchiectasis; the persistent low grade temperature, weight loss, and listlessness, with or without marked cough, that hangs on after an acute respiratory infection, may be the first vague hint of developing bronchiectasis. In any suspected case, bronchography should be done. If there is any suspicion of atelectasis from a mucus plug, growth, or pressure from outside the bronchus, a bronchoscopy should be done.

The treatment of advanced bronchiectasis is surgical removal of the affected areas if this can still be done; lobectomy, lingulectomy and other segmental operations, or even pneumonectomy occasionally are the surgical methods in use. The operation of lobectomy twenty-five years ago, carried a mortality rate of about 50 to 60 per cent, in general. In 1940, Churchill[25] reported a series of 124 lobectomies, with a 2.4 per cent mortality. Others reported

no mortality from this operation during recent years. Churchill and Belsey[24] gave impetus to the tissue saving operation of segmental resection, particularly as it applied to the lingular segment. Graham[26] reported the successful removal of both lower lobes, the right middle lobe, and the lingula of the left upper lobe, in one patient.

The treatment of bronchiectasis that is too advanced for surgery is palliative: postural and bronchoscopic drainage; penicillin, sulpha drugs, and streptomycin by parenteral or aerosol administration; or sulfa drugs by mouth are all helpful during the acute episodes; vaccines (stock and especially autogenous); general hygenic measures as proper nutrition, including adequate protein and vitamines; attention to the cardiac complications; residence in a warm, dry climate are all useful. Climate itself may have an ameliorating influence on bronchiectasis. The prompt treatment of acute or chronic sinusitis and tonsilitis cannot be over emphasized; and in spite of the pernicious tendency of many people to overuse vasoconstricting nasal drops until the mucus membranes become swollen and boggy with paralized vasoconstricting muscles, the judicious, limited use of vasoconstrictors, in drops or by local application with a cotton tipped applicator, in sinusitis is as well indicated as is incision and drainage for an abscess elsewhere in the body. Also, removal of bony or hyperplastic obstructions in the nose are necessary.

The proper use of vaccines in early bronchiectasis often gives surprisingly good results at all ages, but especially in children. In children, the regenerative power is superior, and a defect in a child will be relatively smaller when the part has reached adult growth. In using vaccines, it will sometimes be found that the stock vaccine contains antigens closely enough related to the patient's own pathogens that an autogenous vaccine may not be necessary; but more often an autogenous vaccine made from the patient's own sputum becomes preferable.

There are several points in the use of vaccines in bronchiectasis that may make the difference between success or failure; in the first place, these treatments must be kept up for long periods of time, several years at least; however, after a time, such as a year, the periods between doses, which may have been one week, may be lengthened to two weeks or even a month; and rest periods of a couple of months may be taken. The administration should be started with doses small enough to avoid large general and local reactions.

While allergy to such extrinsic agents as foods, epidermals, or pollens, often precedes infection in the respiratory tract and acts by preparing the tract for chronic sinusitis, bronchitis, or bron-

chiectasis, there are a lesser number of patients in whom the intrinsic infection seems to act as a basic sensitizer, making the patient more susceptible to the extrinsic allergens; and when this happens, the patient may be treated for years with extrinsic allergens without results until the intrinsic bacteral antigen is added. The asthma that develops in middle age after an acute respiratory infection and for which no extrinsic allergenic cause can be found, supports this explanation.

Over twenty years ago, while I was practicing in Colorado Springs, Colorado, the chest specialists there tried out pneumothorax and phrenic paralysis for bronchiectasis; at the same time, these methods were tested extensively elsewhere. Our results were very poor, probably mainly because we chose old, advanced cases with hard, thick, unyielding scar tissue around the dilated bronchi. On the other hand, Hennell[27] reported excellent results recently in four cases of early bronchiectasis by using pneumothorax. This is understandable if the disease is early enough; the collapse squeezes out the infective secretions; it stops the rythmical, respiratory pull on the infection-weakened bronchial walls; it approximates the walls of the infected lung units, and smaller bronchi, which contain little or no cartilage, and thus reduces space for secretions to collect as well as aids fibrotic obliteration; it produces a relative anoxia which inhibits the growth of aerobes; and it produces a slowed circulation with its bacteriostatic and local fibrosing effect. Of course, if the bronchial infection were mainly anaerobic, the collapse therapy in bronchiectasis might encourage the growth of the organisms.

However, minor collapse procedures as Banyai's pneumoperitoneum, phrenic paralysis, and pneumothorax have a very important place as emergency treatments for massive pulmonary hemorrhage in bronchiectasis. Probably the most universally useful one of these is pneumoperitoneum: 1) because it is not always possible to determine immediately which lung is bleeding; 2) the basal portion of the lung, the frequent bleeding site, is often uncollapsible by pneumothorax because of pleural adhesions; 3) pneumoperitoneum is usually the least dangerous and most reversible of these procedures.

SUMMARY

The incidence of bronchiectasis is greater than generally realized.

Bronchiectasis runs a progressively downward course from its inception, and usually claims its victims in the third decade. Its serious complications are: heart failure, metastatic abscesses to the brain and other parts of the body, anyloidosis, lung abscess, pleural empyema and attacks of pneumonitis or pneumonia.

Leaving aside congenital bronchal cysts and other developmental malformations, the cause of bronchiectasis is respiratory infection; stenosis of the bronchi; lack of drainage; infection of the bronchial walls with weakening and destruction of the elastic supporting elements; the rythmical inspiratory pull on the weakened walls, which may be augmented by atelectasis or a check-valve mechanism, which increase the stretching effect on the walls.

The best treatment for bronchiectasis is prevention or treatment of the early phase: removal of a plug of mucus causing atelectasis following bronchial or virus pneumonia or other respiratory infections; prompt treatment of a subacute or chronic bronchitis; prompt treatment of a sinusitis and tonsilitis; attention to respiratory allergies; removal of extrinsic foreign bodies; treatment for endobronchial tumors, extrabronchial tumors and enlarged tracheobronchial and hilar glands; drugs such as sulpha drugs or antibiotics; general hygiene and good nutrition; vaccines (autogenous or occasionally stock) and sometimes a warm, dry climate.

The treatment for advanced bronchiectasis is mainly surgical, if the patient has not advanced to a terminal stage. This consists of lobectomy, segmental resection, and occasionally pneumonectomy. The younger patient with his superior regenerative power and his greater anatomical and physiological reserves is the preferred surgical risk in operations for bronchiectasis.

RESUMEN

La frecuencia de la bronquiectasia es mayor de lo que generalmente se supone.

Desde sus principios la bronquiectasia prosigue un curso progresivamente descendiente y, por lo general, reclama sus víctimas en la tercera década. Sus complicaciones graves son: insuficiencia cardíaca, abscesos metastásticos al cerebro o a otras partes del cuerpo, amiloidosis, absceso pulmonar, empiema pleural y ataques de neumonitis o neumonia.

Pasando por alto los quistes bronquiales congénitos y otras anomalías del desarrollo, la causa de la bronquiectasia es la infección respiratoria; la estenosis de los bronquios; la falta de canalización; la infección de las paredes bronquiales con debilitamiento y destrucción de los elementos elásticos de soporte; la tensión respiratoria rítmica sobre las paredes debilitadas, lo que puede ser aumentado por atelectasia o un mecanismo de válvula de retención que aumenta el efecto de la tensión sobre las paredes.

El mejor tratamiento de la bronquiectasia es la profilaxia, o sea, el tratamiento de la fase temprana: la extracción de un tapón de moco que cause atelectasia subsiguiente a una bronconeumonía o neumonía de virus o a otras infecciones respira-

torias; el tratamiento oportuno de la bronquitis subaguda o crónica; el tratamiento oportuno de senositis y tonsilitis; el tratamiento de las alergias respiratorias; la extracción de cuerpos extraños extrínsecos; el tratamiento de tumores endobronquiales o extrabronquiales y de ganglios tráqueobronquiales e hiliares hipertrofiados; el empleo de drogas, tales como las sulfonamidas o los antibióticos; la higiene general y la buena alimentación; las vacunas (autógenas u, ocasionalmente, no autógenas) y, a veces, un clima cálido y seco.

El tratamiento de la bronquiectasia avanzada es principalmente quirúrgico, si el paciente no ha avanzado a un estado terminal. Este tratamiento consiste de lobectomía, resección segmentaria y, ocasionalmente neumonectomía. El paciente más joven, con su superior poder regenerativo y sus mayores reservas anatómicas y fisiológicas es el riesgo quirúrgico preferido en operaciones para la bronquiectasia.

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A Case of Aspergillosis of the Lung

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Aspergillosis of the lung is a very rare disease and is due chiefly to the Aspergillus fumigatus, but sometimes to A. niger, and, even more rarely, to the Aspergillus flavus. This disease is usually confused with pulmonary tuberculosis, and, in fact, sometimes co-exists with it. Because there are on record very few such cases not co-existent with pulmonary tuberculosis, we have thought it desirable to describe our case of aspergillosis of the lung, which for a long time was treated as pulmonary tuberculosis.

L.P., 45 years of age, a civil servant. Between 1941 and 1944 he had mild but frequent hemoptyses which subsided without treatment and which were followed by a light paroxysmal cough without expectoration. In 1945 he noticed general weakness of the body, slight loss of weight, a slight evening rise in temperature, an increase in cough with either a great deal of expectoration of pus and mucus or expectoration of blood, as well as such nervous disturbances as insomnia, numbness of the limbs, and undue fatigue. At this time the diagnosis of pulmonary tuberculosis was made and the patient was offered appropriate treatment.

However, in spite of the clinical and x-ray findings, his sputum was consistently negative for acid-fast bacilli on simple smears and concentrates. The patient remained at rest for many months. But in spite of this general treatment, he failed to improve and on April 15, 1946, he came to us for examination.

During the examination of the patient, we were impressed by the characteristic expectoration containing many small, hard, yellowish or whitish grains of varying sizes, and the sputum being consistently negative for tubercle bacilli. Sputum cultures revealed Aspergillus fumigatus.

The spores of these fungi are usually found spread on the ground, in the dust of dry leaves and in chaff, and reach the lungs by inhalation. These spores are usually not dangerous. They take on a pathogenic character, however, in cases of lowered resistance, such as, during convalescence after serious communicable diseases, and during chronic illnesses such as diabetes, etc. They are occasionally found as secondary invaders in bronchiectasis, chronic bronchitis, and pulmonary tuberculosis. The disease usually appears in those who are occupied in breeding pigeons (in fact, in bird-breeders in general), and in those whose occupation necessitates their coming into contact with the flour of rye.

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Aspergillosis of the lung can be either primary or secondary. The primary form appears as bronchial or bronchopneumonic. Aspergillar bronchitis is manifested at first by a persistent and paroxysmal cough, and is later followed by a great deal of expectoration; it is also sometimes the cause of bronchial asthma. When the fungus is carried through the bronchi and is located in the lung tissue, the disease then takes the bronchopneumonic form and is followed, in addition to the above symptoms, by frequent hemoptyses. The sputum has a characteristic appearance because in it appear a great many whitish or yellowish granules due to necrosis of the lung tissue. Aspergillosis is usually confused with pulmonary tuberculosis because it is located in the upper lobes

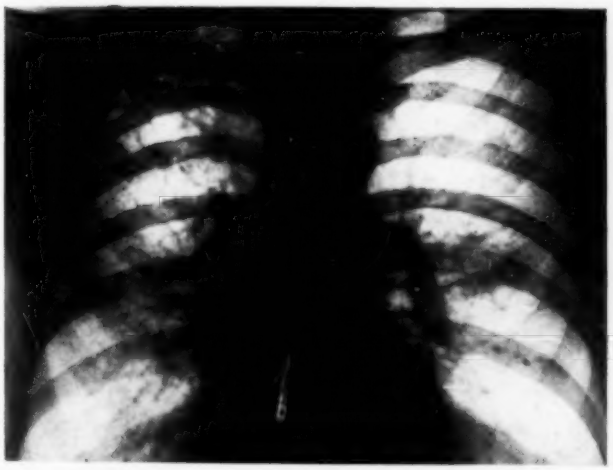

FIGURE 1: Infiltrating lesions in the right lung and the left lower lobe due to Aspergillosis.

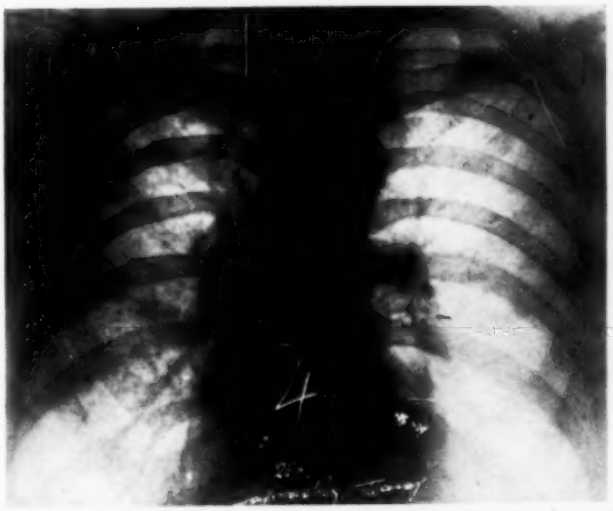

FIGURE 2: The same case after four months treatment showing some resolution of the infiltrations.

and apices. But the clinical picture of aspergillosis of the lung appears much less serious than some forms of pulmonary tuberculosis because in its first stages it usually develops without fever and does not influence the general condition of the patient. However, in its later stages it brings about considerable lowering of the general condition. Differential diagnosis is possible only through the discovery of the pathogenic factor in the sputum.

Pathologically, aspergillosis of the lung causes necrotic areas of the mucosa and of the lung tissue, of a gray or green-gray color, which are surrounded by other cellular elements that are in different stages of the degeneration. Around these areas, the lung shows chronic infiltration, more or less diffuse. The necrotic areas usually communicate with the lumens of the bronchi, and necrotic tissue is expelled by cough and cavities are formed which differ from gangrene only in the absence of foetid breath. This chronic inflammation of the lungs can spread out and contribute to the hardening of the whole of one or both lungs.

In certain cases, in spite of the presence of small cavities, there are no symptoms. Usually, however, there appear such symptoms (similar to those of pulmonary tuberculosis of chronic bronchitis) as cough with pus and mucus, and, more usually, bloody expectoration with, sometimes, the expulsion of the necrotic lung tissue as well.

The disease can sometimes, but only very rarely, heal spontaneously, leaving behind a general lung cirrhosis.

In our case, we applied the treatment of sulfathiazole, giving by mouth four grams daily in three-hourly doses, and adding some sodium bicarbonate. We continued this treatment for four months, except for some slight interruptions, and gave a total of 420 grams of sulfathiazole which the patient tolerated well. Simultaneously, the patient was taking small quantities of potassium iodide which had to be abandoned after the first month because he was unable to tolerate this. After the second month the expectoration decreased considerably and the sputum changed steadily into mucus, and all the necrotic elements and blood disappeared. Sputum cultures have been negative, and the patient has been in an excellent condition for more than a year. He has returned to work and no longer has any complaints.

SUMMARY

A case of pulmonary aspergillosis treated as pulmonary tuberculosis for many years has been discussed. The character of the sputum and the persistantly negative examination for tubercle bacilli led to repeated cultures of the sputum with the aspergillus

fumigatus finally being recovered. Treatment with sulfathiazole and potassium iodide has produced complete cure within 5 months.

RESUMEN

Se discute un caso de aspergilosis pulmonar que fue tratado como tuberculosis pulmonar por muchos años. El carácter del esputo y el hecho de que la busca de bacilos tuberculosos siempre resultó negativa, necesitaron que se hicieran repetidos cultivos del esputo, hasta que al fin se encontró el aspergillus fumigatus. El tratamiento con el sulfatiazol y el yoduro de potasio produjo una curación completa en menos de cinco meses.

Atelectasis of the Right Upper Lobe

J. TANCA MARENGO, M.D., F.C.C.P.,* and
JULIO MATA MARTINEZ, M.D., F.C.C.P.**
Guayaquil, Ecuador

The case that we are going to present, according to its principal complication, atelectasis, contains nothing of particular interest, as these cases become every day more easily recognized and diagnosed. However certain circumstances of its development, the indefiniteness of its etiology, and above all the radiologic changes observed in different phases suggested a diagnosis of carcinoma of the lung, which fortunately was subsequently found to be incorrect.

R.L., 52: European refugee, electrician, consulted us for the first time on the 28th of November 1946, with the following complaints—wheezing cough, tenacious muco-purulent expectoration, moderate dyspnea, aggravated by strain and loss of strength, weight and appetite. These symptoms began in May of the same year but in a mild form that did not keep the patient from work. In July he consulted a doctor and at this time his first x-ray film was taken, on the basis of which bed rest was recommended. The patient did not follow the advice, thinking it unnecessary. As his symptoms were not so severe as to hamper his work, he succeeded in staying up and about until in November the cough and dyspnea increased, obliging him to cut down his activities.

At this time we examined the patient noting among other things, exhaustion and debility and his labored respiration even while in a sitting position. Examination of his circulatory system showed little; pulse, 90, regular as to rate, rhythm and intensity; auscultation of heart, negative; blood pressure, 115/70. The respiratory apparatus was carefully examined, with the findings of pharyngitis, imbedded infected tonsils with grade 2 hypertrophy, rhinopharyngitis, and a slight muco-purulent postnasal drip The right maxillary sinus was somewhat opaque on x-ray examination but puncture revealed nothing. The examination of the lung showed equal amplitude in the respiratory movements; the percussion note was resonant throughout, except in the upper third of the right thorax where impairment was noted. Auscultation showed increased breath sounds and sibilant rales and rhonchi in both lungs and, in the area of dullness in the right lung, subcrepitant rales. The temperature was 37° C.; weight 128 lbs.; urine, clear, amber, negative for albumen, sugar and bile pigments.

The patient said he had never been seriously ill, and in the nine years since he came to Ecuador has suffered only an occasional cold and coryza. He insisted that he had never had any respiratory disease either here or in Europe and reiterated several times, confirmed by his wife

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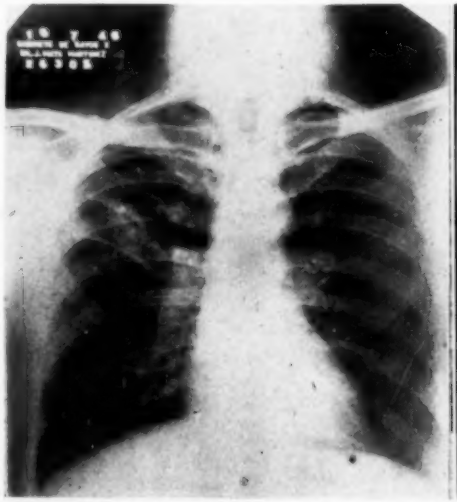

FIGURE 1

FIGURE 2

FIGURE 3

Fig. 1 (July 16, 1946): Standard roentgenogram of the thorax. Emphasized upper right interlobar fissure. Opacity above the fissure, which gives the impression of being a parenchymal infiltration. In the central portion of this opacity one may observe an elliptical shadow which suggests cavity. Inference: tuberculous infiltration with cavity.—*Fig. 2 (December 19, 1946):* Marked opacity which occupies the apex and the interclaviculohilar region on the right, having in its inferior limit the form of a double line which passes from above downward and from without inward. This opacity gives the appearance corresponding to stalectasis of the upper lobe which has drawn the interlobar fissure upward and medialward. In association with this the trachea is drawn slightly to the right. In the remaining part of the right lung field one may observe areas of disseminate infiltration which alternate with radiolucent areas which suggest emplysematous bullae. The left lung field presents no pathological change.—*Fig. 3 (December 19, 1946):* Bucky film. The limits of the opacity in the superior lobe may be seen more clearly. Numerous small radiolucent areas are seen suggesting emphysema.

who was present, that he cannot recall ever having had an illness which confined him to bed. He had never heard of any asthma or similar disease being present in his family. These assurances seemed important since it is rare to find a primary asthma starting in a patient of over 50, and we concluded that the respiratory syndrome was secondary to some lesion in the right apex, first thinking of course of tuberculosis as a possibility which seemed to be confirmed by the first x-ray film, taken in July. X-ray films No. 2 and 3 taken on December 19 somewhat confused the picture and we enlarged the scope of possible etiology to three major diseases: early carcinoma, tuberculosis, atypical pneumonia, the latter having developed in the last few days, secondary to some other unknown disease.

Meanwhile the patient's condition became worse, streaks of fresh blood were found in his sputum and on December 5 he began to run an irregular remittent fever which persisted the 24 days he was hospitalized. During this time the following examinations were made: Hemogram and blood count, showing leukocytosis of 12,000 with normal differential count. Sputum, muco-purulent in appearance, tenacious, slight in quantity but many specimens containing blood, sometimes fresh, sometimes old. Eight specimens were examined on different days, including fresh specimen and homogenized and cultured specimens and only pneumococci Freidlander's bacillus and streptococci were found. No protozoa, mycelia, cells undergoing mitotic changes, or acid-fast bacilli were detected.

The patient was treated with sulfadiazine and penicillin up to 1,800,000 units during which treatment his temperature dropped and his cough,

FIGURE 4

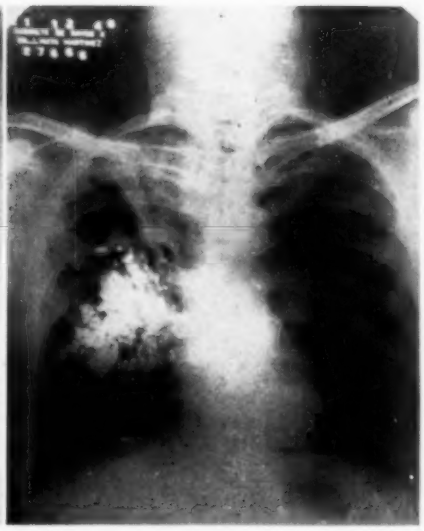

FIGURE 5

Fig. 4 (December 21, 1946): Right Bronchogram. One observes a block about 3 cm. from the beginning of the right superior lobe bronchus. The middle and inferior lobe bronchi are patent. One sees characteristic shadows of bronchiectasis at the level of the tertiary branch bronchi of the middle lobe. *Fig. 5 (December 21, 1946):* Roentgenogram after bronchogram. Iodized oil has not penetrated the superior lobe bronchus. One sees the bronchial pattern alveoli corresponding to the other bronchial branches and in some of these, dilatations may be seen.

expectoration and other pulmonary symptoms diminished. Owing to the position of the shadow high in the lung, bronchoscopy would not have given us much information, and we decided rather to do a bronchogram. The bronchus to the right upper lobe was obstructed and the obstruction ended conically (X-ray film No. 4) which misled us seriously, since it is generally accepted that this is the usual picture in bronchogenic carcinoma. Shortly thereafter film No. 5 was taken to see if it was possible to do a bronchial aspiration, but the x-ray film showed that the obstruction was complete.

Ten days later another bronchogram (X-ray film No. 6) was done and on studying it we found evidence that contradicted this former serious diagnosis, since the blockage of the bronchus had not persisted and the atalectasis had decreased appreciably. The patient's condition improved progressively, the fever ebbed, the cough diminished, the sputum volume decreased, and appetite and strength returned so that he left the clinic the first of January. He was seen every 15 days to affirm that all the symptoms, objective and subjective, had completely disappeared. X-ray film No. 7, taken on April 10, showed a normal thorax. Weight was 134 lbs., that is higher than when we first saw him. He had already resumed his work without experiencing any of his former discomforts.

The third bronchogram taken April 23 (X-ray films No. 8 and 9) showed bronchial dilatation in the right upper and middle lobes, but the respiratory dynamics were established sufficiently to permit the iodized oil to pass into the three tertiary bronchi of the upper lobe. The guinea

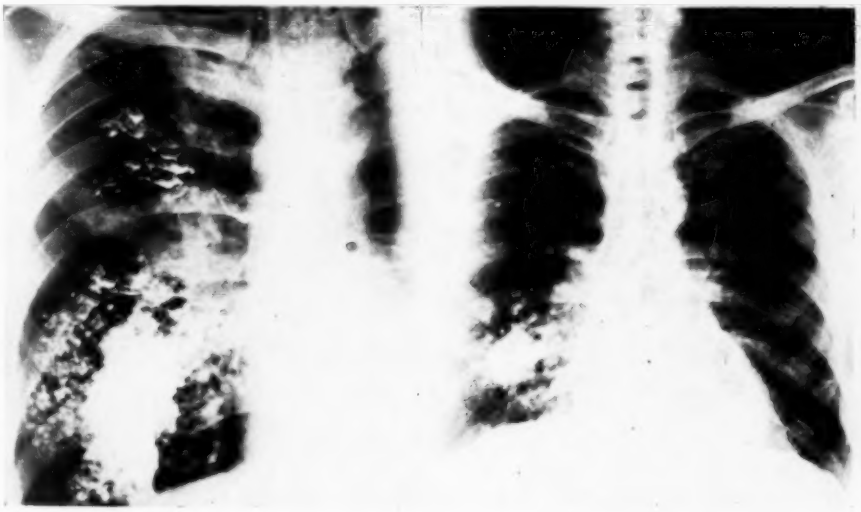

FIGURE 6 FIGURE 7

Fig. 6 (December 31, 1946): Second bronchogram. The bronchial obstruction in the upper lobe has disappeared coincidently with a manifest diminution in the opacity. Iodized oil has penetrated the axillary and anterior branches of the upper lobe but not the apical branch which corresponds to the area where opacity persists. The position of the right superior lobe bronchus has descended to normal. In these films one sees the right middle and inferior lobe bronchi and their branches. The right main bronchus and the trachea are outlined by the contrast medium. One can see the normal bronchial pattern and several dilated tertiary branch bronchi.—*Fig. 7 (April 4, 1947):* Both pulmonary fields appear clear. One can see some as yet unexpelled remnants of iodized oil in the right lower lung.

pigs which had been inoculated were killed at the end of the eighth week and showed no pathological change in any of their organs.

Discussion: The symptomatology did not present a true picture in this disease. The physical and laboratory findings were not specifically informative and allowed one only to infer the existence of an inflammatory focus in the right apex, accompanied by bilateral bronchial asthma. The etiological differentiation rested essentially on the x-ray studies done during the course of the disease and confirmed by the clinical findings and additional laboratory information.

Of the radiological evidence, the most important finding was the atelectasis of the right upper lobe, produced by the obstruction of the bronchus to the right upper lobe, as seen in the bronchogram. The atelectasis may have been produced by the following causes:

(1) Instrinsic bronchial obstruction due to—

a) mechanical action by a foreign body or a mucuous plug.

b) endobronchial inflammatory process. For example, tuberculous granulation of the mucous membrane that occludes the bronchus and ulcerations that, invading the bronchial wall, causes scar tissue, which leads to an obstruction.

Furthermore we must not forget that the conditions mentioned act not only by their mere existence but also

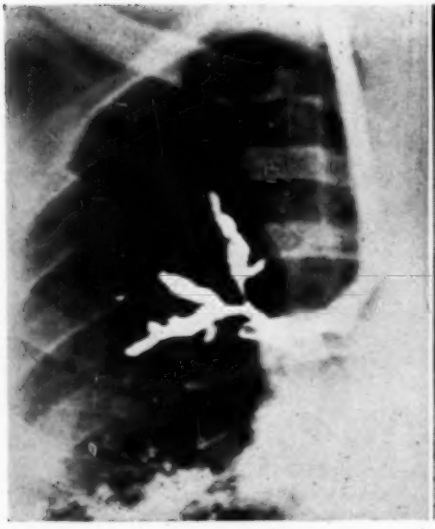

FIGURE 8

FIGURE 9

Fig. 8 (April 23, 1947): Third bronchogram. The three branches of the right upper lobe bronchus are seen by means of filling with the contrast medium. *Fig. 9 (April 23, 1947):* One can see the three branches of the right upper lobe bronchus and their subdivisions, in which some bronchial dilatation appears. One can also see the branches of the middle and lower lobe bronchi, the former also with some dilatations.

principally in an indirect way, by setting up an inflammation of the mucosa.

c) bronchogenic tumor which obstructs as it grows.

(2) External pressure produced by a process generally inflammatory or neoplastic. The density of the shadow in the lobe and the conical occlusion that was seen in the first bronchogram indicate why we had at first considered the possibility of a bronchogenic carcinoma of the right lung. The continued loss of weight and the blood-streaked sputa seemed to support this, but the rapid restoration of the patency of the bronchus shown in the second bronchogram resolved this doubt.

It seems to us that the chronological evolution of this case can be interpreted as follows: The patient had, unknown to him, a chronic infection of the upper respiratory system as shown by the post-nasal drip and the opacity of the right maxillary sinus. We suppose that at the beginning of his acute illness some mucopurulent plugs from these sites or some locally formed inflammatory products obstructed the bronchus to the right upper lobe. There is evidence of this in the first x-ray film, in which the axillary branch of the bronchus seems affected which gave the false impression of a cavity with perifocal reaction. This radiographic picture may now be construed as emphysema and partial atalectasis. These pathological conditions are enough to explain the asthma-like dyspnea which the patient had suffered for five months before we saw him. We suppose in addition that during this long period the bronchial obstruction may have been reversible as well as variable in regards to its position.

In the first days of December the development of the obstruction of the trunk of the bronchus to the right upper lobe established the atalectasis of the entire lobe and the consequent infectious process. The etiological factors were numerous and some of them were definitely identified: pneumococci, Friedlander's bacilli, and streptococci. The last bronchogram on April 23 confirmed the complete patency of the bronchus and showed a residual bronchiectasis.

SUMMARY

The authors present a case of penumopathy of a subchronic type, which became suddenly acute. The x-ray films, in which an extensive atalectasis of the right upper lobe was seen, suggested several possible diagnoses, among which were bronchogenic carcinoma and tuberculosis. The subsequent development of the acute process eliminated these and pointed clearly to the diagnosis of atalectasis due to bronchial obstruction of an inflammatory origin beginning in a chronic infection of the nasal sinuses.

RESUMEN

Los autores presentan un caso de neumopatía de marcha subcrónica que se agudizó súbitamente. Las imágenes radiográficas, en las que se destacaba una atelectasia masiva del lóbulo superior derecho, sugirieron lógicamente varios diagnósticos presuntivos entre los que primaban los de carcinoma broncogenético y tuberculosis. La evolución de la enfermedad eliminó estas hipótesis y permitió definir el diagnóstico de atelectasia por obstrucción bronquial intrínseca de origen inflamatorio determinada por infección crónica de los senos paranasales.

The By-Laws

ARTICLE I

Name

In conformity with its charter this society shall be known as the

AMERICAN COLLEGE OF CHEST PHYSICIANS

ARTICLE II

Objectives

The objectives and purposes of the College shall be:

(a) to establish a world society composed of qualified physicians of high standing who devote all or a major portion of their time to the treatment, study or teaching of, or to research in, diseases of the chest or related specialties;

(b) to encourage and aid medical colleges in establishing a systematic method of teaching diseases of the chest so as to equip the medical student with a knowledge of the fundamentals which have to do with the technic and importance of early diagnosis, the technic and importance of early intensive treatment and the technic and importance of the prevention and control of all diseases of the chest;

(c) to enhance and maintain the interest of practicing physicians in diseases of the chest and to further their training in this specialty;

(d) to maintain and advance the highest possible standards in medical education, medical practice and research pertaining to diseases of the chest;

(e) to maintain high standards of specialization among chest physicians, and

(f) to promote the public welfare in connection with the specialty of diseases of the chest.

ARTICLE III

Membership

Section 1: Members of the College shall be of the following classes: Fellows, Fellows Emeritus, Associate Fellows, Associate Members and Honorary Members. Fellows shall be entitled to all the privileges of the College including the privilege of voting and holding office. Other members shall holding office. Only Fellows and Fellows Emeritus shall be privileged to use the letters "F.C.C.P." after their names. Other members shall be entitled to all the privileges of the College except the privilege of voting and the privilege of holding office.

Section 2: A roll of all members of the College shall be kept by the Executive Secretary of the College.

Section 3: Qualifications of Fellows and Associate Fellows: Every applicant for membership as a Fellow and Associate Fellow shall be prepared to meet the following general requirements:

(a) He shall be the possessor of a diploma conferring the degree of Doctor of Medicine upon him from a medical school acceptable to the Board of Regents.

(b) He shall be a member in good standing of his local, state, and national medical societies, provided, however, that if he is

(1) a full time teacher or research worker in a hospital or institution acceptable to the Board of Regents;

(2) a medical officer in the service of the United States Government, or

(3) engaged solely in the public welfare service,

the requirement of membership in a local, state or national medical society may be waived by the Governor, Regent and Chairman of the Board of Regents upon a consideration of his application, as provided in Section 7 of this Article III.

(c) He shall be prepared to furnish a written statement setting forth his qualifications and fitness for becoming a Fellow or Associate Fellow of the College, as the case may be.

In addition, every applicant for membership as a Fellow shall be prepared to meet the following requirements:

(a) He shall be more than 29 years of age;

(b) He shall have received his degree of Doctor of Medicine at least 5 years prior to his election to the College;

(c) He shall have had at least 5 years' training in the specialty of Diseases of the Chest.

(d) He shall be prepared to take a qualification examination to be given from time to time, as the Board of Regents may direct;

and every applicant for membership as an Associate Fellow shall be prepared to meet the following requirements:

(a) He shall be more than 25 years of age;

(b) He shall have received his degree of Doctor of Medicine at least 2 years prior to his election to the College;

(c) He shall have had at least 2 years' training in the specialty of Diseases of the Chest.

Section 4: Qualifications of Fellows Emeritus: Every Fellow Emeritus shall have attained the age of 65 years and shall have been a Fellow of the College at least 15 years immediately prior to his designation as a Fellow Emeritus. However, the Board of Regents shall have power to designate such other Fellows as Fellows Emeritus as the Board of Regents may deem proper. Fellows Emeritus shall enjoy all the privileges of the College except the privilege of voting and the privilege of holding office. Should they desire the official publication of the College, they shall pay the regular subscription price.

Section 5: Qualifications of Associate Members: Every Associate Member shall have met the same requirements as an Associate Fellow except that his work need not consist of such specialties as are required of Associate Fellows but may, at the discretion of the Board of Regents, simply relate thereto.

Section 6: Qualifications of Honorary Members: The Board of Regents may grant Honorary Membership to such qualified physicians as they may deem to be worthy thereof, in accordance with such rules and regulations as they may from time to time prescribe.

Section 7: Mode of Admission:

(a) An applicant for membership as a Fellow, Associate Fellow, or Associate Member of the College shall

(1) prepare and submit an application setting forth his qualifications as a Fellow, Associate Fellow or Associate Member, as the case may be, on an application form which may be secured from the office of the Executive Secretary of the College, and

(2) deliver the application, together with his dues for the current year and, in the case of a Fellow, his Fellowship fee, and in the case of an Associate Fellow, partial payment of his Fellowship fee, to the Executive Secretary of the College.

(b) If the application is in proper form, the Executive Secretary of the College shall give notice thereof to the Fellows of the College within the metropolitan area in which the applicant resides, or, if the applicant resides in a rural area, to the Fellows of the College within a radius of approximately 100 miles of the place of residence of the applicant, regardless of political boundaries, and shall request them to state the reason or reasons, if any, why the application should not be favorably acted upon.

(c) Two weeks after the mailing of the notice to such Fellows, if the applicant resides in the United States, the Executive Secretary of the College shall forward the application to the Governor for the state wherein the applicant resides, together with a summary of any comments or remarks concerning the applicant submitted by any Fellow of the College.

(d) The Governor shall either approve or disapprove the application. If he disapproves it, he shall send the application to the Regent for the district in which the applicant resides, setting forth the reasons for his disapproval. If he approves it, he shall affix his signature on the application in the space provided for thereon, and shall forward it to the Regent for the district in which the applicant resides.

(e) The Regent shall either approve or disapprove the application.

(1) If he disapproves it, he shall return it to the Governor of the College for the state wherein the applicant resides, setting forth the reasons for his disapproval. If he approves it, he shall affix his signature on the application in the space provided for thereon and shall forward it

to the Chairman of the Board of Regents.

(2) In the event that the Governor and Regent are unable to agree on the approval or disapproval of the application, both the Governor and the Regent shall file a report with the Chairman of the Board of Regents, setting forth their reasons for approving or disapproving the application, as the case may be.

(f) The Chairman of the Board of Regents shall either approve or disapprove the application, and shall present all of the documents to the Board of Regents for final action at their next meeting. The applicant shall be notified of his rejection only after due consideration and final action by the Board of Regents at its next immediate regular meeting.

(g) If the applicant is seeking admission as an Associate Fellow or Associate Member and the Chairman of the Board of Regents approves the application, the Chairman shall affix his signature to the application in the space provided for thereon and return it to the Executive Office of the College. The Executive Secretary shall then notify the applicant of his admission.

(h) If the applicant is seeking admission as a Fellow and the Chairman of the Board of Regents approves the application, the Chairman shall affix his signature on the application in the space provided for thereon, and shall return it to the Executive Office of the College, and, unless the requirement of examination be waived by the Board of Regents, shall direct the Executive Secretary to notify the applicant to appear and take a qualification examination at a time and place specified by the Board of Regents.

(i) (1) If the applicant passes his examination, or if the requirement of examination be waived by the Board of Regents, the Chairman of the Board of Regents shall so notify the Executive Office of the College in writing, and the Executive Secretary shall thereupon inform the applicant that he has been admitted as a Fellow of the College. If the applicant fails to pass his examination, the requirement of examination not having been waived by the Board of Regents, the Chairman of the Board of Regents shall so notify the applicant and the applicant shall be given the opportunity to take the examination the following year. No candidate shall be permitted to take the examination more than once in a given calendar year and he shall not be given more than three opportunities to pass the examination. In the event that an applicant for Fellowship fails to pass the examination, the Chairman of the Board of Regents shall be authorized to refund without interest 75 per cent of all moneys heretofore paid to the College by the applicant in connection with his application for a Fellowship, the remaining 25 per cent being retained to cover the cost incident to examination. If the applicant is rejected for any other reason, all moneys, without interest, will be refunded.

(2) The rejection of an applicant need not be based upon a mere failure of the applicant to meet the qualifications of Sections 3 or 5 of this Article, as the case may be, but may rest on any ground which those passing upon such application shall deem sufficient. The Board of Regents may from time to time adopt such additional rules and regulations relating to admissions as it may deem advisable, subject to alteration and revision from time to time by the College.

(3) Convocations for confirming certificates of Fellowship of the College shall be held at each annual meeting of the College. All new Fellows shall be required to sign the register of the College at a convocation.

(4) If an applicant seeking admission is affiliated with the medical department of the United States Army, United States Navy, United States

Air Force, Veterans Administration or Public Health Service, his application shall be submitted to the Governor of the College for that respective service for approval. If the application is approved by the Governor of the College for that service, it shall then be forwarded to the Chairman of the Board of Regents for approval. If the application is approved or disapproved by either the Governor or the Chairman of the Board of Regents, the applicant shall be governed by the rules set forth in Article III, Sections 7 and 8 of the By-Laws.

Section 8: An Associate Fellow shall make application for membership as a Fellow within five years after his admission as an Associate Fellow of the College. During such period as he remains an Associate Fellow he shall pay yearly installments toward his Fellowship fee as directed by the Board of Regents of the College. In the event that his application for Fellowship is disapproved, any and all moneys paid to the College toward his Fellowship fee shall be refunded to him in full without interest.

Section 9: Discipline: Any member of the College may be disciplined or expelled for conduct which, in the opinion of the Board of Regents, is unbecoming a Fellow or is derogatory to the dignity of the College or inconsistent with its purposes. The expulsion of a member may only be ordered upon the affirmative vote of two-thirds of the members of the Board of Regents present at a regular or special meeting and only after such member has been informed of the charges against him and has been given an opportunity to present a defense to such charges before the Board of Regents.

ARTICLE IV

Officers

Section 1: The officers of the College shall consist of a President, a President-Elect, a First Vice-President, a Second Vice-President and a Treasurer.

Section 2: The President-Elect, the First Vice-President, the Second Vice-President and the Treasurer shall be elected at the annual meeting of the College, each for a term of one year. The President-Elect shall enter upon his duties as President at the end of the first administrative session of the first annual meeting following his election.

Section 3: The President of the College shall preside at all regular meetings of the College and at all convocations for conferring certificates of Fellowship of the College. He shall appoint within 60 days after his election to office, members of all councils and standing committees and shall from time to time appoint such other committees as may be necessary or convenient to carry on the activities of the organization. He shall name the chairmen of each council and committee. He shall direct the activities of each council and committee and shall be an ex-officio member of each council and committee except the nominating committee.

Section 4: The President-Elect shall keep in close touch with the affairs of the College. He shall be an ex-officio member of all committees.

Section 5: The First Vice-President shall assume the duties of the President in the event of the death, resignation or absence of the President.

Section 6: The Second Vice-President shall preside at all meetings of the College in the absence of the President and the First Vice-President; and in the event of the death, resignation or disability of the President and the First Vice-President, he shall perform the duties and exercise the power of the President.

Section 7: The Treasurer shall sign all checks and other documents dealing with the financial transactions of the College. He shall authorize the deposit of all funds of the College in a bank approved by the Board of Regents and dispense same on checks, signed by him and by the auditor of the College, upon receipt of vouchers signed by the Executive Secretary. In his absence, and for limited periods of time, he may delegate the power of receiving and disbursing the funds of the College to an acting Treasurer appointed by the President. At each annual meeting of the College the Treasurer shall submit to the Board of Regents a detailed statement of the financial condition of the College for the fiscal year ending December 31, approved by a certified public accountant, which shall reflect the amount of moneys he has

received and expended during the past year. He shall furnish with such statement a written report summarizing the statement. He shall further instruct the Executive Secretry to notify the membership at large of the financial condition of the College either by letter or in the official publication of the organization. He shall have access to the safety vaults in the presence of the Executive Secretary or his bonded designated assistant for the inspection, deposit or withdrawal of securities or other valuable property of the College. He shall be bonded in the name of the College for an amount as may be designated by the Board of Regents, for the faithful performance of his trust, the premium of which shall be paid by the College.

ARTICLE V

Board of Regents

Section 1: The Board of Regents shall consist of the Past-Presidents of the College, the President, the President-Elect, the First Vice-President, the Second Vice-President, the Treasurer, the Chairman of the Board of Regents and one Regent elected from each regional district of the College. The Chairman of the Board of Governors for the College shall be an ex-officio member of the Board of Regents with the right to vote. The Executive Secretary or one of his designated assistants shall serve as Secretary to the Board of Regents.

Section 2: One of the members of the Board of Regents shall be elected as its Chairman by a majority vote of the members of the Board of Regents present at an annual meeting and one of the members of the Board of Regents shall be elected as Vice-Chairman by a majority of votes of the members of the Board of Regents present at an annual meeting. The Chairman and the Vice-Chairman shall serve for a period of one year and shall be eligible for re-election at each annual meeting. Should the Chairman be an elected member of the Board, the President shall appoint a Fellow from the regional district of the Chairman, as Regent, to replace the Regent elected as the Chairman of the Board of Regents.

Section 3: The elected members of the Board shall each serve for a term of three years and no member may serve more than three consecutive terms, provided, however, that at the first election of the Board approximately one-third of the elected members shall be elected for a one-year term, approximately one-third for a two-year term, and the remaining one-third for a three-year term. The original division of the members into approximate thirds shall be made by the President of the College.

Section 4: In general, the Board shall be the business managers of the College. Specifically they shall have power to regulate and conserve property interests of the College, to fix the annual dues of members, to adopt from time to time rules and regulations for the election of Fellows and Associates to the College, including the giving of examinations, supplementary to and not conflicting with the regulations contained in these By-Laws, to determine what publications are to be sponsored by the College, to make contracts in connection therewith and to name editorial boards for each publication sponsored, to grant charters to state, territory and district organizations, to divide the states, territories and foreign countries having members in the College into appropriate regional districts and to assign the supervision of each such district to a member of the Board and to transact any and all business not otherwise provided for pertaining to the organization and operation of the College.

Section 5: Ten members of the Board shall constitute a quorum for the transaction of business. Any Regent may be represented by a Fellow of the College in good standing who holds his written proxy and who resides in the regional district represented by such Regent.

Section 6: There shall be an annual meeting of the Board of Regents at the regular annual meeting of the College and, when necessary, a semi-annual meeting to be held at a place designated by the Executive Council. Additional special meetings of the Board may be called by the Chairman or the President upon a written request therefor signed by eleven members of the Board. The Chairman shall preside at all meetings. In his absence the President shall preside.

Section 7: During the interim periods between meetings of the Board, the business affairs of the College shall be managed by an Executive Council composed of the President, the President-Elect, the First Vice-President, the Second Vice-President, the Treasurer, and the Chairman and one elected member of the Board of Re-

gents. The Executive Secretary shall be the Secretary of the Executive Council. The Past-Presidents of the College shall serve as ex-officio members of the Executive Council.

Section 8: Whenever a vacancy may occur among the elected members of the Board, the President shall appoint a Fellow from the regional district represented by the Regent whose place is vacant to fill the vacancy until the next annual meeting of the College, at which time the College shall elect a member to fill the vacancy.

Section 9: The Board may from time to time relocate the states, territories and foreign countries into different regional districts and may increase the number of regional districts.

Section 10: The Board shall appoint an Executive Secretary who shall have supervision of the activities and business affairs of the College. The Executive Secretary, or one of his designated assistants, shall (1) act as Secretary to the Board of Regents, the Executive Council and the Board of Governors; (2) direct the Executive Offices of the College, maintaining therein all records, such as those of membership, inventories and accountings; (3) prepare a budget covering all expenditures of the organization for submission to, and approval of, the Board of Regents; (4) direct the mailing of all notices and other forms to be sent to the membership or to the members of the Board of Regents and the Board of Governors; (5) to coordinate the activities of the various College chapters, councils and committees; and (6) to perform such other duties as may be assigned to him from time to time by the various governing bodies of the College. No agreement entered into by the Board of Regents for the services of an Executive Secretary may be of more than three years' duration.

ARTICLE VI

Board of Governors

Section 1: The Board of Governors of the College shall be composed of members of the College to be elected as provided in Section 3 of this article at each annual meeting of the College as follows: One member shall be elected from each state, territory or possession of the United States which has one or more Fellows of the College; one member shall be elected from each of the following services: The United States Army, the United States Navy, the United States Air Force, the Veterans Administration, Public Health Service and the United States Indian Service; and one member shall be elected from each foreign country which has one or more Fellows of the College. Whenever a vacancy may occur in the membership of the Board of Governors, the President of the College may appoint a Fellow from the same state, territory, possession, medical service or country formerly represented by the Governor whose place is vacant, to fill the vacancy until the next annual meeting of the College.

Section 2: The Board shall elect one of its members to serve as Chairman of the Board for a period of one year and he shall be a member of the Board of Regents. The Chairman may be re-elected for not more than three consecutive terms.

Section 3: The elected members of the Board of Governors shall each serve for a term of three years, provided, however, that at the first election of the Board approximately one-third of the elected members shall be elected for a one-year term, approximately one-third shall be elected for a two-year term, and the remaining one-third shall be elected for a three-year term. Thereafter all members shall be elected for a three-year term. The original division of the members into approximate thirds shall be made by the President of the College.

Section 4: It shall be the duty of the individual Governors to coordinate and further the best interests of the College in their respective states, territories, possessions, medical services or countries, as the case may be, and to perform such other duties as may be assigned to them by the Board of Regents.

Section 5: The Board shall meet in annual session at the annual meeting of the College or at the call of the Chairman with the approval of the President of the College.

Section 6: In the event that a member of the Board is unable to attend the meeting he may be represented by a Fellow of the College in good standing, who holds his written proxy, and who resides within the same state, territory, possession, or country represented by the Governor, or who is a member of the same medical service represented by the Governor, as the case may be.

Section 7: No member of the Board of Governors except its Chairman may at the same time be a member of the Board of Regents.

Section 8: The Executive Secretary of the College, or one of his designated assistants, acting in his behalf, shall serve as Secretary of the Board of Governors.

ARTICLE VII

Fees

Section 1: A Fellowship fee shall be required of all Fellows of the College, subject, however, to the provisions of Section 2 of this Article VII. Annual dues shall be required of all members of the College except Honorary Members and Fellows Emeritus. The amount of the Fellowship fee and of the annual dues of members shall be determined by the Board of Regents.

Section 2: At the discretion of the Board of Regents, and without publicity, any Fellowship fee or annual dues may be waived in whole or in part.

Section 3: Dues shall be payable on January 1 of each year and shall become delinquent if not paid by July 1 of that year. A member who is delinquent in his dues automatically loses all privileges of the College including subscription to the official publication. A member whose dues are delinquent shall be notified of such delinquency by a regular letter, and on September 1 he shall be notified by a registered letter with return receipt requested. The notification shall contain formal notice that the member is delinquent in the payment of his dues and that if the dues are not paid by December 31 of that year, he will be dropped from the rolls of the College. If the dues are not paid by December 31, one year from the time his dues become payable, the member shall be dropped from the rolls by order of the Board of Regents.

Section 4: Any member dropped from membership because of delinquency who desires to re-establish his membership must file a new application therefor and must pay the regular Fellowship fee, if he was a Fellow, and the current dues then required by the Board of Regents, together with all past unpaid dues, unless waived in whole or in part by the Board.

ARTICLE VIII

Publications

Section 1: The Board of Regents, through the Executive Secretary, shall issue from time to time a directory of the College containing the names and addresses of the members of the College and designating those members who are qualified to administer pneumothorax treatments or who are qualified in any related branch of the specialty of diseases of the chest. The Board may also sponsor such other publications as it deems desirable and in the interests of the College.

Section 2: Editorial Boards for publications sponsored by the College shall be composed of Fellows of the College and shall consist of not less than three or more than five members.

Section 3: Each Editorial Board shall have authority to enter into contracts, subject to the approval of the Board of Regents, or to make other arrangements for the production and distribution of its publications, and shall have the power to elect associate editors to its staff from the members of the College.

Section 4: The editorial policy of each publication shall be in accord with the objectives of the College. A majority of the members of an Editorial Board shall be required to approve the publication of any material in its publication which any member of such Board considers a matter of policy. Each Editorial Board shall be responsible to the Board of Regents of the College.

Section 5: The Editorial Board of the publication which is designated by the Board of Regents as the official organ of the College shall be required to publish in that journal a summary of the transactions of the annual meetings of the College and of the meetings of the Board of Regents.

Section 6: The Board of Regents shall elect the members of the Editorial Board and provide a term of office for each member not to exceed three years, and shall also provide that the terms of each member of the board be staggered. Any member of an Editorial Board who has served three years may be re-elected for three years upon the recommendation of the other members of the Editorial Board. Any new members elected to the Editorial Board should first receive approval of the members of the Editorial Board. Each Editorial Board shall elect from among its own mem-

bers a Chairman who shall act as Editor-in-Chief of its publication.

ARTICLE IX

Meetings

Section 1: An annual meeting of the College shall be held each year, immediately preceding or immediately following the meeting of the American Medical Association, as may be designated by the Board of Regents and in the same city as that meeting, for the election of officers and for the transaction of such other business as may be necessary.

(a) The Board of Regents may, if desirable, authorize the Executive Secretary of the College to select a city other than the city in which the American Medical Association meets, for the holding of annual meetings of the College.

Section 2: The College shall conduct scientific sessions at each annual meeting, which shall be devoted to diseases of the chest. The scientific sessions shall be open to all legally licensed physicians, graduate and undergraduate students of medicine.

Section 3: The President shall appoint committees to assist in the arrangements for the annual meetings.

ARTICLE X

Councils and Committees

Section 1: All councils and committees of the College shall be appointed by the President, who shall designate a chairman, vice-chairman and secretary of each council and committee. Members of councils and committees shall be both individually and collectively responsible to the Board of Regents for the conduct of their respective organizations. Reports and other publications of all councils and committees shall be approved by the Board of Regents before clearance for publication.

Section 2: Each council and committee shall be empowered to further to their best ability the purposes for which it has been established, in accordance with the objects of the College. All councils and committees shall be required to render a report concerning their activities at the annual meeting of the College, or as otherwise directed by the President.

Section 3: The Chairman of each council and committee, unless exempted from this duty by the President, shall be required to furnish to the President an outline of the projects to be carried out by his council or committee within 90 days after he has accepted the Chairmanship of such council or committee.

Section 4: (a) The following councils, consisting of nine members each, shall be appointed:

Council on Undergraduate Medical Education,
Council on Postgraduate Medical Education,
Council on Public Health,
Council of Tuberculosis Hospitals,
Council of Tuberculosis Committees,
Council on the Management and Treatment of Diseases of the Chest,
Council on Public Relations.

At the first appointment of these councils three of such members shall be appointed for a one-year term, three for a two-year term, and three for a three-year term. Every year thereafter three members shall be appointed for a three-year term.

(b) The number of members of each of the following councils shall be determined by the President. Each member shall serve for a one-year term:

Council on International Affairs,
Council on Pan American Affairs,
Council on European Affairs,
Council on Pan Pacific Affairs,
Council on African and Near East Affairs.

(c) The Council on Research shall be composed of two sections, the Financial Section and the Scientific Section.

(1) The Financial Section shall consist of nine members, each member to serve for a period of three years and the terms of office shall be staggered so that three new members are appointed each year by the President with the advice and consent of the Board of Regents. The President shall appoint or re-appoint as chairman one of the senior members of the Section. The duties of the Financial Section shall be to raise funds to carry on the work of the council and to secure potentially available endowments and grants.

(2) The Scientific Section shall consist of five members whose terms of office shall be staggered so that one member

will serve for five years, one for four years, one for three years, one for two years, and one for one year, and that one member shall be appointed each year thereafter by the President for a term of five years, with the advice and consent of the Board of Regents. The President shall appoint a chairman of the Scientific Section with the approval of the Board of Regents. The duties of the Scientific Section shall be to consider and approve research projects to be undertaken by the College. Their recommendations shall be approved by the Council on Research before being presented to the Board of Regents.

(3) There shall be a general chairman of the Council to coordinate the activities of the Financial Section and the Scientific Section and he shall be appointed by the President with the approval of the Board of Regents.

(d) All policies, programs and expenditures recommended by these councils must be approved by the Board of Regents.

Section 5: (a) There shall be a standing Committee on Constitution and By-Laws composed of five members appointed by the President. At the first appointment of this committee, one member shall be appointed for a five-year term, one for a four-year term, one for a three-year term, one for a two-year term, and one for a one-year term, and thereafter one member shall be appointed each year for a term of five years. Members shall be eligible for re-appointment.

(b) The duties of the Committee on Constitution and By-Laws shall be to consider all proposed changes for amendments to the Constitution and By-Laws and to study and propose amendments from time to time as changing conditions may demand. These recommendations shall be referred to the Board of Regents for approval.

Section 6: The number of members of each of the following committees shall be determined by the President. Each member shall serve for a one-year term and may be re-appointed:

Committee on Membership,
Committee on College Awards,
Committee on Scientific Program.

Section 7: The chairmen of councils and standing committees shall appoint such sub-committees to serve under the jurisdiction of the council or committee, as the case may be, as may be deemed advisable or as directed by the Board of Regents of the College. These committees may be discharged by the President or the Board of Regents of the College after they have served their purpose. All appointments to sub-committees shall be for a period of one year unless the sub-committee be sooner discharged.

Section 8: There shall also be a Nominating Committee consisting of three Fellows of the College. One member shall be a member of the Board of Regents and he shall be elected by the members of the Board of Regents at their annual meeting. One member shall be a member of the Board of Governors and he shall be elected by the members of the Board of Governors at their annual meeting. The third member of the committee shall be a Fellow at large and he shall be appointed by the President and shall serve as chairman. The Nominating Committee shall nominate the following officers:

(1) President-Elect,
(2) First Vice-President,
(3) Second Vice-President,
(4) Treasurer,
(5) Historian,
(6) Regents whose terms expired during the current year, and
(7) Governors whose terms expired during the current year.

Nominations by the Nominating Committee shall not preclude nominations from the floor at the elections of the various officers.

Section 9: The President shall appoint a Board of Examiners to consist of three or more members. Each member shall serve for a one-year term. The President shall appoint a chairman, vice-chairman and secretary from among the members of this board. Members of this board may be re-appointed. The Board of Examiners may call upon any Fellow of the College to assist in examinations.

ARTICLE XI

Chapters

Section 1: State and district chapters of the College may be formed and shall apply to the Board of Regents for a charter. Charters shall be issued upon the following conditions:

(a) That all the members of the state or district chapter be duly elected members of the College and in good standing;

(b) That at least one meeting of such state or district chapter be held at the same city, and on or about the same date, as the annual meeting of the state or district medical society in organized medicine covering the same geographical area, or in connection with a recognized medical school or postgraduate assembly;

(c) That a minimum of fifteen Fellows, Associate Fellows or Associate Members in any one state, group of states, or territory, constituting a recognized medical district, be enrolled as members of the chapter for the purpose of electing officers and conducting the business of the chapter. All officers shall be elected for a period of one year;

(d) That the name to be used by such state or district chapter shall be approved by the Board of Regents of the College;

(e) That the By-Laws of each state or district chapter be approved by the Board of Regents of the College;

(f) That the state or district chapter be self-supporting;

(g) That the five immediate past-presidents of the chapter be constituted as an Executive Committee. In inaugurating the Executive Committee, the past-president of the chapter shall serve as chairman until the Executive Committee is comprised of five past-presidents. Thereafter each senior past-president shall serve as the chairman of the Executive Committee, in the proper order, for a period of one year, and each additional past-president shall serve on the Executive Committee for a period of five years. The President, Vice-President, and Secretary-Treasurer of the chapter shall be ex-officio members of the Executive Committee. The activities of the chapter shall be directed by the officers and members of the Executive Committee. It shall be the duty of the Executive Committee to approve the policy of the chapter and to coordinate its activities. The Executive Committee shall guide the officers of the chapter in the proper performance of the duties and responsibilities.

Charters shall be granted by the Board of Regents upon receipt of a written application therefor, which shall satisfy the foregoing conditions, signed by the duly elected officers of the state or district chapter and directed to the Chairman of the Board of Regents of the College.

Section 2: Action for the withdrawal of the charter of any state or district chapter may be initiated for cause upon written complaint filed by a Fellow of the College with the chairman of the Board of Regents. Before any charter can be withdrawn the officers, if known, of the state or district chapter shall be given written notice of the charges filed against such chapter and shall be given opportunity to appear at a meeting of the Board of Regents, designated in the notice, to be held not sooner than four weeks after the mailing of such notice and shall be permitted to submit testimony and evidence at such meeting as to why such charter should not be withdrawn. If the officers of the chapter are not known to the Board then such notice shall be given to all known Fellows of the College in that state or district.

ARTICLE XII

Membership in Countries Other Than the United States of America

Section 1: Objective: It shall be the purpose of the College to extend its objectives into all countries of the world.

Section 2: Qualifications for Membership: Applicants for membership in countries other than the United States of America shall be classified as Fellows and Associate Members. In countries outside of the United States of America the qualifications for Fellowship and Associate Membership shall be such as the Board of Regents shall establish. Both Fellows and Associate Members in countries other than the United States of America shall be entitled to all the privileges of the College, including the privilege of voting and holding office.

Section 3: Mode of Admission:

(a) The applicant for membership as a Fellow or Associate Member of the College shall:

(1) prepare and submit an application setting forth his qualifications as a Fellow or Associate Member, as the case may be, on an application form which may be secured from the office of the Executive Secretary of the College, and,

(2) deliver the application, together with his dues for the current year and, in the case of a Fellow, his Fellowship fee, to the Secretary-Treasurer of the chapter of the College in the country wherein the applicant resides, or, if there is no chapter, to the Executive Secretary of the College.

(a) The Fellowship fee and dues of each chapter shall be approved by the Board of Regents.

(b) In countries where monetary restrictions exist, the money shall be deposited in a bank approved by the Board of Regents to the credit of the American College of Chest Physicians. This money may be withdrawn only on the order of the Treasurer of the College.

(3) It is the intent of the Board of Regents of the College that examining boards be set up as early as possible in each country and whenever feasible. The examining board shall be chosen by the Board of Regents from the members of the chapter in that country.

(b) The application shall be approved by the Governor for the country or the district wherein the applicant resides and if there is a Regent for the country, or district, the application shall also be approved by the Regent and then submitted to the Chairman of the Board of Regents for final approval. If there is neither a Governor nor a Regent in the country or the district wherein the applicant resides, the application is to be sent to the Executive Secretary of the College who shall forward the application to the Chairman of the Board of Regents for approval.

(c) In the event that the Governor and Regent are unable to agree on the approval or disapproval of the application, both the Governor and the Regent shall file a report with the Chairman of the Board of Regents, setting forth their reasons for approving or disapproving the application, as the case may be.

Section 4: Discipline: Any member of the College may be disciplined or expelled for conduct which in the opinion of the Board of Regents is unbecoming a Fellow or is derogatory to the dignity of the College or inconsistent with its purposes. The expulsion of a member may only be ordered upon the affirmative vote of two-thirds of the members of the Board of Regents present at a regular or special meeting and only after such member has been informed of the charges against him and has been given an opportunity to present a defense to such charges before the Board of Regents.

Section 5: Governors: Each country where there are one or more members of the College may have a Governor. Governors shall be nominated and elected at the annual meetings of the College and shall serve for a term of one year. The duties of the Governors shall be the same as set forth in Article VI of these By-Laws, with the exception of Section 3.

Section 6: Regents: In countries having College chapters, the members of the chapter may petition the Board of Regents of the College for the election of a Regent. Should the petition be granted, such Regents shall be nominated and elected at the annual meetings of the College and they shall serve for a term of one year. The duties of such Regents shall be the same as set forth in Article V of these By-Laws with the exception of Section 3.

Section 7: Fees: The Fellowship fees and membership dues in countries other than the United States of America shall be determined by the Board of Regents.

Section 8: Charters:

(a) Chapters of the College may be formed in any country and officials of such chapters shall apply to the Board of Regents of

the College for charters. Charters to such chapters shall be issued upon the following conditions:

(1) That all members of the chapter de duly elected members of the College in good standing;

(2) That at least one meeting of the chapter be held each year for the purpose of conducting a scientific session and electing officers, in addition to conducting all necessary business of the chapter. All officers shall be elected for a period of one year;

(3) That a minimum of 15 Fellows and/or Associate Members for any one country, state, province, or group of countries, states, and/or provinces, be enrolled as members of such national or district chapters;

(4) That the name to be used by chapters shall be approved by the Board of Regents of the College;

(5) That the By-Laws of each chapter be approved by the Board of Regents of the College;

(6) That the chapters be self-supporting;

(7) That the five immediate past-presidents of the chapter be constituted as an Executive Committee. In inaugurating the Executive Committee, the past-presidents of the chapter shall serve as chairman until the Executive Committee is comprised of five past-presidents. Thereafter each senior past-president shall serve as the chairman of the Executive Committee, in the proper order, for a period of one year, and each additional past-president shall serve on the Executive Committee for a period of five years. The President, Vice-President, and Secretary-Treasurer of the chapter shall be ex-officio members of the Executive Committee. The activities of the chapter shall be directed by the officers and members of the Executive Committee. It shall be the duty of the Executive Committee to approve the policy of the chapter and to coordinate its activities. The Executive Committee shall guide the officers of the chapter in the proper performance of the duties and responsibilities.

Charters may be granted by the Board of Regents upon receipt of a written application therefor, which shall satisfy the foregoing conditions, signed by the duly elected officers of such chapters and directed to the Chairman of the Board of Regents of the College.

(b) The charter of any country or group of countries having a chapter may be suspended or withdrawn for cause upon written complaint filed by a Fellow of the College with the Chairman of the Board of Regents, setting forth the reasons for requesting such withdrawal. Before any charter can be withdrawn the officers, if known, of the chapter shall be given written notice of the charges filed against such chapter and shall be given an opportunity to appear at a meeting of the Board of Regents, designated in the notice, to be held not sooner than four weeks after the mailing of such notice and shall be permitted to submit testimony and evidence at such meeting as to why such charter should not be withdrawn. If the officers of the chapter are not known to the Board then such notice shall be given to all known Fellows of the College in that country or group of countries.

(c) All members and officials of College chapters shall be responsible for the conduct of their activities to the Council on International Affairs of the College and shall make annual reports to the Council concerning the activities of the College in their respective countries.

ARTICLE XIII

Amendments

Any Fellow of the College in good standing may initiate a proposal for an amendment to these By-Laws. Such proposal shall be presented in writing to the Executive Secretary of the College, who shall submit it to the Committee on Constitution and By-Laws for consideration and report to the Board of Regents at their next meeting. The Board of Regents shall then

vote on the advisability of presenting the proposed amendment to the Fellowship as a whole for its consideration. A majority of the members of the Board of Regents present, voting favorably for any proposed change in the By-Laws, shall make it mandatory upon the Executive Secretary to notify the Fellowship at large, either by letter or through the official publication of the College, of the text of the proposed amendment. This notification must be presented to the Fellowship at least sixty days before the annual meeting. The Executive Secretary shall then present the proposed amendment to the Fellowship of the College at the annual meeting for their adoption or rejection. A two-third vote of the Fellows present and voting shall be necessary to adopt an amendment.

ARTICLE XIV

Original Members and Officers

The members of this corporation shall consist of the members of the American College of Chest Physicians, an unincorporated association, and such other members as may from time to time hereafter be admitted to membership in accordance with the provisions of these By-Laws. The officers, Regents, Governors, committee members and members of the editorial boards of this corporation shall be the officers, Regents, Governors, committee members, and members of the editorial boards of the unincorporated association until such time as their term of office or membership would have expired had the unincorporated association continued in existence.

ARTICLE XV

The rules contained in "Robert's Rules of Order, Revised Edition" shall govern the American College of Chest Physicians in all cases to which they are applicable and in which they are not inconsistent with the existing by-laws.

COMMITTEE ON COLLEGE BY-LAWS

Charles M. Hendricks, M.D., El Paso, Texas, Chairman
Carl H. Gellenthien, M.D., Valmora, New Mexico
Minas Joannides, M.D., Chicago, Illinois
Hollis E. Johnson, M.D., Nashville, Tennessee
Walter E. Vest, M.D., Huntington, West Virginia

At the 13th Annual Meeting of the American College of Chest Physicians held at the Ambassador Hotel, Atlantic City, New Jersey, June 5-8, 1947, a resolution was adopted by the Board of Regents requesting the President to appoint a Committee on College By-Laws. The above committee was appointed by Major General S. U. Marietta, President of the College at that time, and the committee was authorized to study the present by-laws and recommend revisions in order to bring the by-laws up to date.

The first meeting of the committee was held at the Hotel Statler, Washington, D. C., on November 21, 1947, at which time the first draft of the by-laws was studied by the committee. The second meeting of the committee was held at the Congress Hotel, Chicago, Illinois, on June 17, 1948, and a second draft of the revised by-laws was studied. The final draft of the revised by-laws was submitted to the Board of Regents of the College at their semi-annual meeting in Miami Beach, Florida, on October 23, 1948. The Board of Regents of the College approved the by-laws with several minor revisions.

In accord with Article 12 of the College By-Laws, the revised by-laws as approved by the Board of Regents of the College have been published herein and they will be submitted for final approval to the Fellowship of the College at the Administrative Session to be held at the Ambassador Hotel, Atlantic City, New Jersey, on June 4, 1949.

College Chapter News

FIFTEENTH ANNUAL MEETING
AMERICAN COLLEGE OF CHEST PHYSICIANS

The Fifteenth Annual Meeting of the American College of Chest Physicians will be held at the Ambassador Hotel, Atlantic City, New Jersey, June 2 through 5, 1949, just prior to the annual meeting of the American Medical Association, June 6 through 10. Members are urged to make their hotel reservations at once and may use the form appearing on page xi of this issue for that purpose. A limited number of rooms are available at the Ambassador Hotel for members wishing to remain for both meetings.

MISSOURI CHAPTER

The Missouri Chapter of the College held a most successful dinner meeting and scientific session at the Chase Hotel, St. Louis, on November 29, just prior to the opening of the Interim Session of the American Medical Association held in St. Louis. Guest speaker at the dinner was George Saslow, M.D., St. Louis, who spoke on "Psychotherapy in the Treatment of Pulmonary Tuberculosis."

Alfred Goldman, M.D., F.C.C.P., St. Louis, Governor of the College for Missouri, presided at the dinner and introduced George D. Kettelkamp, M.D., F.C.C.P., Koch, President of the Chapter. Mr. Murray Kornfeld, Chicago, Executive Secretary of the College, was introduced and presented a brief talk on the activities of the College.

A number of College Fellows who are Delegates in the House of Delegates of the American Medical Association were present in St. Louis and attended the chapter meeting. They were: Walter E. Vest, M.D., F.C.C.P., Huntington, West Virginia; Carl H. Gellenthien, M.D., F.C.C.P., Valmora, New Mexico; Robert H. Hayes, M.D., F.C.C.P., Chicago, Illinois; and Bernard Klein, M.D., F.C.C.P., Joliet, Illinois. Also present were: Gerald Beatty, M.D., F.C.C.P., Wilmington, Governor of the College for Delaware; Hugh L. Houston, M.D., F.C.C.P., Murray, Kentucky, President-Elect of the Kentucky State Medical Society; W. Edwin Bird, M.D., Wilmington, Editor of the Delaware State Medical Journal; and Walter S. Broker, M.D., F.C.C.P., of East St. Louis, Illinois.

A. J. Steiner, M.D., F.C.C.P., St. Louis, Secretary of the Missouri Chapter, and Alfred Goldman, M.D., St. Louis, were in charge of arrangements for the meeting.

After the dinner meeting the following scientific program was presented:

"Suppurative Diseases of the Lungs,"
Minas Joannides, M.D., F.C.C.P., Chicago, Illinois.

"The Effect of Bronchial Infection on Pulmonary Function,"
Edwin R. Levine, M.D., F.C.C.P., Chicago, Illinois.

"Pulmonary Mobilization by Decortication,"
Tom H. Burford, M.D., St. Louis, Missouri.

ACTIVITIES OF COLLEGE MEMBERS IN NEW YORK STATE

The New York State Medical Society will meet in annual session at the Hotel Statler, Buffalo, New York, May 2-5. The annual meeting of the New York State Chapter of the College will meet in conjunction with the meeting of the state medical society.

Nelson W. Strohm, M.D., F.C.C.P., Buffalo, Regent of the College, is Vice-Speaker of the House of Delegates of the New York State Medical Society, and Frederic W. Holcomb, M.D., F.C.C.P., Kingston, is a member of the Council and President of the Third District Medical Society of New York State. Foster Murray, M.D., F.C.C.P., Brooklyn, is Chairman of the Section on Chest Diseases in the state medical society, Samuel A. Thompson, M.D., F.C.C.P., New York City, serves as Secretary, and Grant Thorburn, M.D., F.C.C.P., New York City, President of the New York State Chapter of the College, is Delegate from the Section to the House of Delegates of the state medical society.

Articles by College Fellows recently published in the New York State Journal of Medicine are "Bacterial Filtrates and Autogenous Vaccines in the Treatment of Chronic Sinus Disease and Other Chronic Respiratory Affections," by Hugh M. Kinghorn, M.D., F.C.C.P. and George E. Wilson, M.D., F.C.C.P., Saranac Lake, and "Physical and Roentgenologic Findings in the Early Diagnosis of Nonopaque Foreign Bodies of the Bronchial Tract," by Arthur Q. Penta, M.D., F.C.C.P., Schenectady.

At a meeting of the Saranac Lake Medical Society on November 24, George W. Wright, M.D., F.C.C.P. and R. S. Mitchell, M.D., discussed "Pneumoperitoneum."

The next postgraduate course on diseases of the chest to be given in New York City under the sponsorship of the Council on Postgraduate Medical Education of the College will be held at the Hotel New Yorker, November 14-19, 1949.

For further information concerning the annual meeting of the New York State Chapter of the College please communicate with Donald R. McKay, M.D., F.C.C.P., Secretary of the Chapter, 333 Linwood Avenue, Buffalo, New York.

The present officers of the New York State Chapter are: Grant Thorburn, M.D., New York City, President; Roger A. Hemphill, M.D., Mount Morris, First Vice-President; Joseph J. Witt, M.D., Utica, Second Vice-President; and Donald R. McKay, M.D., Buffalo, Secretary-Treasurer. George Foster Herben, M.D., F.C.C.P., Yonkers, is Governor of the College for New York State.

ORGANIZATION OF THE NORTHEAST BRAZILIAN CHAPTER

On November 19, 1948, in Recife, Brazil, the 37th Chapter of the American College of Chest Physicians was charted as the Northeast Brazilian Chapter. Officials of the College who were present at the organization meeting were: Lopo de Carvalho, M.D., F.C.C.P., Governor of the College for Portugal; Eduardo T. Etzel, M.D., F.C.C.P., Governor for the Southern Brazilian Chapter; Reginaldo Fernandes, M.D., F.C.C.P., Governor for the Central Brazilian Chapter; Jose Silveira, M.D., F.C.C.P., Governor for the East Brazilian Chapter and Affonso MacDowell Filho, M.D., F.C.C.P., Secretary of the Central Brazilian Chapter.

The following officers were elected for the newly organized Northeast Brazilian Chapter:

Joaquim Cavalcanti, M.D., Governor.
Agenor de Sousa Bomfim, M.D., President.
Joao Asfora, M.D., Vice President, State of Pernambuco.
Lourival Moura Dantas, M.D., Vice President, State of Paraiba.
Milton Ribeiro Dantas, M.D., Vice President, State of Rio Grande do Norte.
Otavio Lobo, M.D., Vice President, State of Ceará.
Herodoto Pinheiro Ramos, M.D., Secretary.
Manuel Gomes, M.D., Treasurer.

Among the guests who presented papers at this meeting, which was held in conjunction with the IV National Congress of Tuberculosis, were: Felice Parodi, M.D., of Italy; Roger Even, M.D., of France and Lopo de Caryalho, M.D., F.C.C.P., of Portugal.

Northeast Brazilian Chapter Organizational Meeting
November 19, 1948, Recife, Brazil

Left to right: Reginaldo Fernandez, M.D., F.C.C.P., Governor of the Central Brazilian Chapter; Lopo de Carvalho, M.D., F.C.C.P., Governor for Portugal; Pinheiro Guimaraes, M.D.; Affonso MacDowell Filho, M.D., F.C.C.P., Secretary of the Central Brazilian Chapter and Roger Even, M.D., Guest Speaker, Paris, France.

CENTRAL AMERICAN CHAPTER

The Central American Chapter held its II Annual Reunion at the Sanatorio Antituberculoso, on January 19, 1949, in conjunction with the III Central American Congress of Tuberculosis. Enrique Coronado Iturbide, M.D., F.C.C.P., Governor of the College for Central America,

is also the President of the III Central American Congress of Tuberculosis. Rafael Leal h., M.D., and J. Mauricio Gutiérrez de León, M.D., President and Secretary-Treasurer of the Central American Chapter of the American College of Chest Physicians, respectively, comprise the Board of Directors of the III Central American Congress of Tuberculosis.

PERUVIAN CHAPTER

The Peruvian Chapter of the American College of Chest Physicians held its IV Annual Reunion at the Central Antituberculosis Dispensary in Lima, Peru, during the 20-23 of December, 1948. The following scientific program was presented:

December 20, 1948, 7 P. M.:

"El Instituto de la Tuberculosis,"
Ovidio Garcia Rosell, M.D., F.C.C.P.

"Investigación Tuberculino-Radiológica en los postulantes a la Universidad Mayor de San Marcos, 1947-1948,"
Juan A. Werner, M.D., F.C.C.P.

"Tuberculosis y Ocupación,"
Luis E. Hubner, M.D., F.C.C.P.

"Indice de infección y morbilidad tuberculosa en Magdalena Nueva,"
Manuel Agurto, M.D. y Tomás Cortez, M.D.

December 21, 1948, 7 P. M.:

"Cierre Cavitario por perforación en el curso del nuemotórax terapéutico,"
Luis Cano Gironda, M.D., F.C.C.P.

"La Estreptomicina en el tratamiento de la laringitis tuberculosa,"
Leopoldo Molinari, M.D., F.C.C.P.

"Formas gangliopulmonares y estreptomicina,"
Mario Pastor, M.D., F.C.C.P.

"Tuberculosis gangliopulmonar,"
Victor Narváez, M.D.

"El lavado bronquial en las bronquiectasis,"
Federico Vargas Jiménez, M.D.

December 22, 1948, 7 P. M.:

"O.M. 32 en Tuberculosis,"
Juan A. Werner, M.D., F.C.C.P.

"Lesiones pulmonares no visibles a la radiografía simple,"
Juan Escudero Villar, M.D., F.C.C.P.

"Resultado de las pruebas funcionales y electrocardiográficas en un grupo de operados de toracoplastía,"
Victor Tejada, M.D., F.C.C.P.

"Sarcoma del pulmón,"
Carlos Alberto López, M.D., F.C.C.P.

December 23, 1948, 7 P. M.:

"Tumores broncopulmonares primitivos,"
Max Espinoza Galarza, M.D., F.C.C.P.

"Síndrome toráxico-abdominal producido por alteraciones diafragmáticas,"
Ramón Vargas Machuca, M.D., F.C.C.P.

"La dihidroestreptomicina,"
Leopoldo Molinari, M.D., F.C.C.P.

MEETING, ILLINOIS CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS
Congress Hotel, Chicago, January 7, 1949

Some of the physicians who were present at the dinner given in honor of Dr. Gumersindo Sayago, Cordoba, Argentina.

After the scientific meeting, the following officers were elected for the coming year:

Ramón Vargas Machuca, M.D., Lima, Peru, President.
Juan Escudero Villar, M.D., Lima, Peru, Vice President.
Leopoldo Molinari, M.D., Lima, Peru, Secretary.
Luis E. Hubner, M.D., Callao, Peru, Treasurer.
Luis A. Morales, M.D., Lima, Peru, Librarian.

ILLINOIS CHAPTER

A joint meeting of the Chicago Tuberculosis Society and the Illinois Chapter of the College was held in Chicago at the Congress Hotel on Friday evening, January 7th. A dinner honoring Professor Gumersindo Sayago, F.C.C.P., Cordoba, Argentina, was given just prior to the scientific session. Dr. Sayago was guest speaker at the dinner.

The following scientific program was presented after the dinner meeting:

"Clinical Application of Dihydrostreptomycin,"
David T. Carr, M.D., Department of Medicine, Mayo Clinic.

"Paraminosalicylic Acid in Experimental and Clinical Tuberculosis,"
Robert H. Ebert, M.D., University of Chicago.

"Use of Paraminosalicylic Acid in the Treatment of Tuberculosis,"
Henry C. Sweany, M.D., F.C.C.P., Medical Director of Research, Municipal Tuberculosis Sanitarium.

General Discussion.

DOCTOR GUMERSINDO SAYAGO VISITS THE UNITED STATES OF AMERICA

Dr. Gumersindo Sayago of Cordoba, Argentina, Regent of the American College of Chest Physicians, and Past President of the Argentine Chapter of the College has visited in the United States after completing a tour of the Philippine Islands for the Mission of the Unitarian Service Committee and the World Health Organization of the United Nations. Dr. Sayago was the expert on tuberculosis in a four man commission which was concerned with general health problems in the Philippines. Following completion of the work of the mission, Dr. Sayago remained in the Philippines as an official guest of the Philippine Government to institute an intensive training course on BCG vaccination, bacteriological diagnosis and tuberculin testing for physicians and nurses. While in the Philippine Islands, Dr. Sayago was honored by being awarded the Medal of Rizal as an Honorary Knight of the Rizal Legion. Dr. Sayago also organized the Philippine Chapter of the American College of Chest Physicians and installed the officers of the newly established chapter during his visit.

On December 30 Dr. Sayago arrived in San Francisco where he was met by Dr. Harry C. Warren, Second Vice-President of the College, Dr. Seymour M. Farber, Secretary-Treasurer of the California Chapter of the College, Dr. Sidney Shipman, a Fellow of the College, and Dr. George Becker of the San Francisco Public Health Department.

Dr. Sayago left San Francisco for Minneapolis, arriving there on

January 4, where he was the guest of Dr. Jay Arthur Myers, Editor of the College journal, "Diseases of the Chest." While in Minneapolis, he delivered a lecture before a joint meeting of the Minnesota Chapter of the College and the Minnesota Trudeau Society. Dr. Sumner Cohen, President of the Minnesota Chapter, presided at the meeting.

On January 7 Dr. Sayago delivered a lecture in Chicago on his mission to the Philippine Islands at a joint meeting of the Illinois Chapter of the College and the Chicago Tuberculosis Society. He was introduced at the dinner meeting by Dr. Henry C. Sweany, a member of the Editorial Board of the College journal. During Dr. Sayago's stay in Chicago, he spoke at a staff meeting at the Municipal Tuberculosis Sanitarium and also visited the Tice Clinic for BCG of which Dr. Sol Roy Rosenthal is director.

After leaving Chicago Dr. Sayago visited the University of Michigan at Ann Arbor and then proceeded to Boston where he was the guest of Dr. Richard H. Overholt, President of the College, and Dr. Marcio M. Bueno, Medical Director of the Tuberculosis Division, Fall River General Hospital.

Dr. Sayago's next visit was to New York City where a number of meetings were arranged. While in New York City he was the guest of Dr. Juan R. Herradora, Secretary of the Council on Pan American Affairs of the College. On January 20 Dr. Sayago delivered a lecture at the Henry Phipps Institute, Philadelphia. During his visit there Dr. Sayago was the guest of Dr. Esmond R. Long, Editor of the American Review of Tuberculosis.

On January 20 Dr. Sayago left New York City for Mexico City to attend the VIII Congress of Latin American Tuberculosis Societies (ULAST). Dr. Sayago is a former President of this Society, having presided at the V Congress which was held in Argentina in 1940.

During Dr. Sayago's visit to the United States, he was entertained by many of his friends.

Staff of Tuberculosis Hospital, Nichols Field, Philippine Islands, taken during the visit of Dr. Gumersindo Sayago.

College News Notes

Richard H. Overholt, M.D., F.C.C.P., Brookline, Massachusetts, President of the American College of Chest Physicians, will give a lecture on "Surgery's Place in the Treatment of Pulmonary Tuberculosis" on January 18, at Houston, Texas. The lecture will be given at a meeting honoring Dr. Elva A. Wright. John Roberts Phillips, M.D., F.C.C.P., Houston, will introduce Dr. Overholt. On the following evening Dr. Overholt will speak at a meeting of the Harris County Medical Society and his subject will be "Cancer of the Lung."

Edgar Mayer, M.D., F.C.C.P., New York, N. Y., Chairman of the Council on African and Near East Affairs of the College, is making a trip to the near east for the World Health Organization. He will visit Turkey, Palestine, Egypt, Lebanon, Iraq, Iran and Syria.

A. Barklie Coulter, M.D., F.C.C.P., Washington, D. C., was elected President of the Metropolitan Washington Tuberculosis Conference at a general meeting on October 29 at Glenn Dale Sanatorium. Dr. Coulter, who is Director of the District Bureau for Tuberculosis, succeeds Dr. V. L. Ellicott, Health Officer for Montgomery County, Maryland.

David D. Feld, M.D., F.C.C.P., former acting medical director of the Los Angeles Sanitarium, has accepted appointment as Medical Director of the Jewish Consumptives Relief Society Sanatorium at Denver, Colo.

On October 15, ground was broken at Mt. Vernon, Illinois for the beginning of construction of the first State-owned tuberculosis hospital for the public in Illinois. The hospital will be known as the Mt. Vernon State Tuberculosis Hospital and will be operated under the direct supervision of the State Department of Public Health.

Edwin R. Levine, M.D., F.C.C.P., Chicago, Illinois, has been appointed representative of the Scientific Exhibit for the Section on Diseases of the Chest of the American Medical Association to assist in the procurement of exhibits for the 98th Annual Session of the American Medical Association to be held in Atlantic City, June 6-10, 1949.

Maurice G. Buckles, M.D., F.C.C.P., Columbus, Ohio, presented an interesting paper on "Recent Developments in Chest Surgery" at the regular monthly meeting of the Cabell County Medical Society, held October 14 at the Hotel Prichard in Huntington.

The article by Robert B. Homan Jr., M.D., F.C.C.P., El Paso, Texas entitled "Tuberculosis and the General Practitioner" has been published in a recent issue of the Texas State Journal of Medicine.

Henry C. Sweany, M.D., F.C.C.P., Medical Director of Research at the City of Chicago Municipal Tuberculosis Sanitarium and a member of the Board of Directors of the Tuberculosis Institute of Chicago and Cook County, was awarded the Dearholt Medal by the Mississippi Valley Conference on Tuberculosis. He was cited for his research on bacteriology and pathology of tuberculosis and associated conditions.

Arthur W. Newitt, M.D., F.C.C.P., formerly head of the Chicago Municipal Tuberculosis Sanitarium, has accepted an appointment as tuberculosis control officer for the state of Michigan.

The first annual Clyde M. Fish Memorial Lecture of the Atlantic County Hospital for Tuberculous Diseases, Clyde M. Fish Memorial, was delivered on November 18, 1948 by Burgess Gordon, M.D., F.C.C.P., Philadelphia, Professor of Medicine, Jefferson Medical College, and Director of the Barton Memorial Division for Chest Diseases, Jefferson Medical College.

The address, entitled "Tuberculosis," was open to the profession and to the public. The meeting was held at the Haddon Hall Hotel, Atlantic City, and was presided by Charles Hyman, M.D., F.C.C.P., Medical Director, Clyde M. Fish Memorial.

The Board of Regents has appointed Professor Maurizio Ascoli, M.D., F.C.C.P., of Palermo as Governor of the College for Italy.

COMMITTEE ON CHEMOTHERAPY AND ANTIBIOTICS

The Committee on Chemotherapy and Antibiotics, which serves under the Council on the Management and Treatment of Diseases of the Chest of the College, recently released a report on "The Use of Streptomycin in Tuberculosis." This report was intended primarily for general practitioners, to keep them informed on the recent developments in the use of streptomycin in tuberculosis. Copies of the report were mailed to state, specialty and other medical journals in the United States for publication. The report has appeared in the following medical journals:

The Journal of the Medical Association of the State of Alabama, December, 1948.
The Journal of the Arkansas Medical Society, November, 1948.
The Connecticut State Medical Journal, November, 1948.
The Journal of the Maine Medical Association, November, 1948.
Medical Annals of the District of Columbia, November, 1948.
The Military Surgeon, December, 1948.
The North Carolina Medical Journal, October, 1948.
The Journal of the Ohio State Medical Society, January, 1949.
The Texas State Journal of Medicine, December, 1948.
The West Virginia Medical Journal, November, 1948.

PARA AMINOSALICYLIC ACID

The Panray Corporation is now marketing PAS (Para Aminosalicylic Acid) to sanatoria, hospitals, and medical research workers.

Though still under investigation, PAS is reported to be a promising chemotherapeutic agent in the treatment of tuberculosis. Steady supplies are now available for immediate delivery.

The company has assembled a complete bibliography of important papers dealing with PAS. These are available on request to interested workers. Write The Panray Corp., 398 Broadway, New York, N. Y.